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UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF NEW YORK
----------x
ROBERT A. FALISE; LOUIS KLEIN, JR.;FRANK MACCIAROLA; and CHRISTIAN E. MARKEY, JR.,as trustees,
Plaintiffs, -against-
THE AMERICAN TOBACCO COMPANY;RJ REYNOLDS TOBACCO COMPANY; B.A.T. INDUSTRIES, PLC; BROWN & WILLIAMSONTOBACCO CORPORATION; PHILIP MORRIS INCORPORATED; LIGGETT, INC,;and LORILLARD TOBACCO COMPANY,
Defendants.----------x
666 Fifth Avenue New York, New York
June 5, 2000
10:17 a.m.

Videotaped Deposition of
Expert Witness, WILLIAM J. NICHOLSON, Ph.D.,
pursuant to Subpoena, before Cindy DiLeonardo,
a Notary Public of the State of New York.

ELLEN GRAUER COURT REPORTING CO. 110 East 59th Street, 25th Floor
New York, New York 10022 212-750-6434
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A P P E A R A N C E S:

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Winston-Salem, North Carolina 27102
BY: THOMAS D. SCHROEDER, ESQ.

ALSO PRESENT:
JIM WOOD, Videographer

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IT IS HEREBY STIPULATED AND AGREED by and between the attorneys for the respective parties herein, that the filing, sealing and certification of the within

deposition be waived.
IT IS FURTHER STIPULATED AND AGREED that all objections, except as to the form of the question, shall be reserved to the time of the trial.
IT IS FURTHER STIPULATED AND AGREED that the within deposition may be sworn to and signed before any officer authorized to administer an oath with the same force and effect as if signed and sworn to before the Court.

- oOo -

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THE VIDEOGRAPHER: Good morning. My name is Jim Wood with Video Documentaries for Ellen Grauer Reporting, New York City.
The time is 10:17 a.m. Today is Monday, June 5, 2000.
Today's videotaped deposition is being taken of William J. Nicholson, Ph.D., in the matter of Falise, et al versus American Tobacco, et al. Case number 99 CV 7392.
Attorneys for the plaintiff are Ms. Laurie S. Dix and Mr. Steven Kazan. Attorney for the defendant is Mr. Thomas Schroeder.
Our reporter is Cindy DiLeonardo. Miss reporter, would you please swear in the witness.

W I L L I A M J. N I C H O L S O N, Ph.D., called as a witness, having been duly sworn by a Notary Public, was examined and testified as follows:

MS. DIX: Before we start I want to note that we produced - as you know,

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we objected to the production of documents for Dr. Nicholson on the ground he was not a testimonial expert witness for us and when the Court addressed this similar issue with Dr. Rubinowitz - when the Court addressed this similar issue in connection with Dr. Rubinowitz he recognized there would likely be work product issues implicated.
What would I would suggest, to make sure we proceed expeditiously and avoid any problems, is that we proceed with the understanding that the

questions be related to the production of Dr. Nicholson's August 30, 1999 report and that any extraneous consulting matters that Dr. Nicholson has undertaken for us would be excluded from your questions.

Obviously if you want to make a record on the other activities that Dr. Nicholson is involved in, that's fine, but I would anticipate that most

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of those I would instruct the witness not to answer on the ground it's work product and that Dr. Nicholson is a consulting expert for those purposes.

MR. SCHROEDER: I think the way to proceed is to go question by question and see what presents itself.

Obviously I disagree with the analysis there is any work product. He has been tendered as an expert with an expert report, which was served in the case. I don't know that we'll cross that threshold, so rather than argue about it now, I propose let's go question by question.

My position would be that there is no work product protection applicable as far as the questions that I intend to ask him.

Why don't we proceed along those lines and see how we do. Is that all right with you?

MS. DIX: That's fine.

EXAMINATION BY

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MR. SCHROEDER:

Q. Dr. Nicholson, would you give us your full name, please?

A. William J. Nicholson.

Q. Dr. Nicholson, my name is Tom Schroeder. I represent RJ Reynolds Tobacco Company, one of the defendants in this lawsuit.

You've been deposed many times before, correct, sir?

A. Yes.

Q. Approximately how many depositions have you given, ballpark number, sir?

A. 50 -- I'm not sure.

Q. You testified in trials before as well?

A. Yes.

Q. About how many times, sir?

A. 20 times, maybe.

Q. You understand that I'm going to ask you a series of questions today and if at any time you do not understand my question, simply let me know and I will be happy to repeat it for you.

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A. Yes.
Q. And if you answer the questions then we are going to proceed with the understanding that you, of course, understood and heard the question, right?
A. Yes.
Q. Where do you live, sir?
A. [DELETED]

Q. How far is that from [DELETED], sir?
A. I don't know how far from [DELETED]. It's probably --
MS. DIX: Objection.
A. 25 miles. It's eight miles from the [DELETED].
Q. You understand you were listed as an expert for the Johns Manville Trust in this litigation?
A. At one time, yes.
Q. Do you know why you were -- what is your understanding as to why you were withdrawn?
MS. DIX: Objection.

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A. My understanding is that the process that was taken place within that time was led to problems, legal problems or unrelated to the issue, but a different trial was established with a different format and that's - roughly speaking that's my understanding.
I am not completely -- I can't explain the two differences completely.
Q. At what point in time were you informed that you would no longer be tendered as an expert in the case?
A. Some time in the last month.
Q. Just to make sure I understand, then, sir, is it fair to say that your understanding as to why you were withdrawn as an expert is because the approach that the plaintiffs were taking in the case no longer related to your analysis?
A. That's correct. The approach that my work would have been used in was -- was no longer the approach that was being taken in the trial.
Q. And what do you understand, sir,

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as to why that was?
MS. DIX: Objection, asked and answered.
Q. You can answer the question, sir.
A. I don't fully understand that other than it could no longer be -- the

approach could no longer have been brought in the Federal Court system and thus would be limited to a particular state, jurisdiction and that was one reason and that's -- there was another reason, I'm sure, too, but that was one that I understood.

Q. You received a subpoena from one of the law firms for the defendants in this case a few weeks ago, do you remember that, sir?

A. Yes.

MR. SCHROEDER: Mark this as Nicholson 1, please.

I don't know if I have more than two copies. I thought you would be familiar with the subpoena.

MR. KAZAN: Is it just the subpoena or notice and proof of service

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as well?

MR. SCHROEDER: I think it's just the subpoena. It's just the subpoena.

(Nicholson Exhibit 1, Subpoena, marked for identification.)

MR. KAZAN: Which subpoena are you talking about?

MR. SCHROEDER: There is a subpoena dated April 8th.

MR. KAZAN: I'm --

MS. DIX: You have one copy for the witness, then I'll take --

MR. SCHROEDER: There is a copy for the witness and I have a copy. I'm sorry, I do have an extra copy. You are welcome to it.

(Handing.)

MS. DIX: Thank you.

Q. Dr. Nicholson, I'm going to hand you what's been marked as Nicholson 1; do you recognize that, sir as a copy of the subpoena that was served on you for documents within the last 30 days or so?

A. Yes, I do.

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Q. And where did you search to respond to the subpoena?

A. Throughout my office.

Q. Where is your office?

A. At 101st Street between Madison and Fifth Avenue in Manhattan.

Q. Is that in the Mount Sinai Medical Center?

A. Yes.

Q. If we can, I want to make sure then that -- let me ask it this way: Are there any documents you found responsive to Exhibit 1 that you did not serve to the defendants in this case?

MS. DIX: Objection, calls for

legal conclusion.
A. Not to my awareness, that I had.
Q. Yes, yes, so everything that you had that you thought was responsive to the subpoena, you turned over, right?
A. Yes, I did.
Q. You turned them over to the lawyers I take it; is that right?
A. Yes.

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Q. Do you know whether or not any documents were withheld from the -- let me ask it this way:
MR. SCHROEDER: There was no privilege log; are there any documents being withheld from the ones he turned over?
MS. DIX: My letter states everything pretty clearly. You want to go off the record.
MR. SCHROEDER: Rather than go through a million questions, simply, if there is something withheld, I'll try to find out what it is.
If everything he gave you was turned over, then --
MS. DIX: I think you are asking the question to the witness.
MR. SCHROEDER: I'll do it that way.
Q. Doctor, you gave everything to your lawyer including Ms. Dix?
A. In response to this, yes.
Q. Do you know whether or not

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everything you gave to the lawyers was in fact turned over to the defendants?
A. I'm not -- I don't believe so. I saw a note on my August 30th report that it wasn't turned over, because it already had been turned over earlier, so there was some circumstances like that when it was unnecessary to do it again and I mentioned this earlier and we decided the simplest thing for me to do was send everything, rather than me make that judgment.
There were also and I don't know whether these were turned over or not, but there could have been copies of articles that had originated from your side that was supplied to me and I, in turn, in response to any document I had relating to this case, had sent them to Ms. Dix, so what was turned over from what I said, I can't answer, but I believe - at least I know in one circumstance from a note something wasn't, but I'm not totally unaware of anything being turned over that you did not already know about or have.
Q. All right.

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All documents relating to Johns Manville or the Johns Manville Settlement Trust, to your knowledge, were all of those collected and given to Ms. Dix?
A. Yes, they were. Other than some cover sheets on fax transmissions and I may have missed some letters that were just --
Q. Over the years you have had and/or your hospital, Mount Sinai, has had a relationship with the Johns Manville Asbestos Company, right?
MS. DIX: Objection.
A. We did have a relationship particularly in the period 1969 through 1974 and for a period thereafter.
They were in conjunction with the asbestos worker's union supporting the program at Mount Sinai on the exposures and insulation industry and how they might be reduced.
Q. Where are the documents that relate to that period of time, 1969 to '74 maintained at Mount Sinai?
A. I don't know.
Q. Are they maintained in your
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office?
A. Documents that I have are represented by what is colloquial known as the green sheets.
I wrote, including other people's comments, an insert to the Asbestos Worker's Journal, which was published every four months that described the work we were doing within that program.
With respect to, for example, air counts or something that might have been taken there, I have no idea where they are and I must say I didn't look through every page in my office, which is four drawer file cabinets and they were on the floor because of file cabinets and another feet of stacks of paper.
It's possible there might be something from that period of time there, I don't believe so, but I believe the green sheets are well representative of that period of time.
Q. Did you keep a separate file on your correspondence with Johns Manville?
A. No.
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Q. Did you keep a separate file with the correspondence with the Johns Manville Personal Injury Settlement Trust?
A. No.
Q. For purposes of this deposition if I call it the trust, will you and I understand

we are talking about the Manville Trust?
A. Yes.
Q. The plaintiff in this case?
A. Sure.
Q. You produced to us copies of all your previous deposition transcripts; is that right?
A. All that I had. I generally don't get a copy of my deposition or if I do before too long would throw them away. I went through the piles of depositions that I had and provided you with every one that was mine.
Q. Thank you, sir.
There was a study of insulators that was conducted between 1981 and 1983?
A. Yes.
Q. That you were a part of, right?
A. Yes.

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Q. That was a portion of what was known as the Selikoff insulator cohort?
A. That's correct. It was a portion of the 17,800 cohort established in 1967.
Q. Did you produce to us copies of all materials you have relating to that cohort?
A. I have some disks relating to that cohort that were - contained material that was being produced to you otherwise.
Q. Is that in response to the separate subpoena to Mount Sinai?
A. Yes.
Q. To your knowledge, those have been turned over?
A. Yes and I provided to Dr. Levin, who was turning the Mount Sinai material over, those disks that would be relevant to that and aid him in putting together a composite of submission to you that would be inclusive of not only the clinical data, but also mortality data related to that cohort with exclusion of items of a personal nature identifying individuals, so everything relating to the

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2907 and such.
Q. Those are the number of participants?
A. Are the Mount Sinai submission and I would only be duplicating it with what I would have.
Q. Fine, sir.
Just so that we are clear, the 2907 insulators that are a part of that 1981 to '83 cohort and if we call that the Nicholson cohort would that be all right with you?
A. Call it the 2907.
Q. We will call it the 2907.
A. It's not my cohort, although I

16 certainly worked with it.
17 Q. The 2907 have been followed as a
18 part of the larger cohort of 17,800, correct?
19 A. Yes.
20 Q. The 2907 have been studied by
21 numerous scientists and doctors as part of the
22 larger cohort of 17,800, right?
23 A. Well, there are several of us at
24 Mount Sinai working on that material, but I
25
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1 wouldn't call us numerous.
2 Q. But my point is --
3 A. We are several in number, but
4 that's --
5 Q. My point is simply the study of
6 the 2907 that was conducted between 1981 and
7 1983, that data from that study have been used
8 by different people for different purposes to
9 look at the asbestos relationship to disease,
10 right?
11 A. Yes, that's correct.
12 Q. Did you provide to Dr. Levin all
13 different analyses of those 2907 persons that
14 has been conducted by Mount Sinai, to your
15 knowledge?
16 A. No, I did not, because I did not
17 have all of that material. I did not have
18 some of the clinical material.
19 Q. I'm talking now --
20 A. There has been a paper published
21 on asbestosis and its mortality from
22 asbestosis and I don't have any of that.
23 Q. Who has that?
24 A. I believe - well, certainly
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1 Steve Markowitz does and I believe Dr. Levin
2 has it and I believe --
3 Q. Steve Markowitz and Dr. Levin are
4 at Mount Sinai?
5 A. Dr. Markowitz is there part-time.
6 His primary position is with the - a college
7 in Brooklyn.
8 Q. What I'm trying to determine,
9 Doctor, is whether what was turned over to
10 Dr. Levin and then in turn turned over to the
11 defendants was the analysis of the 2907
12 insulators conducted simply by you or whether
13 it included data by other reviewers at
14 Mount Sinai, not just you?
15 MS. DIX: Objection.
16 A. I believe all of the data on X-ray
17 abnormalities, pulmonary function and clinical
18 information has either been turned over in
19 disk form and is being produced in hard copy
20 by Infinite Xeroxing.
21 The use that Dr. Markowitz made of
22 that is in the published literature. There is
23 a publication by him on his analysis of
24 asbestosis in this group.

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Q. Other reviewers have used that data and drawn other inferences from the same data, right?
MS. DIX: Objection. What do you mean by that? You are assuming --
MR. SCHROEDER: The last page and-a-half or two pages of questions are simply trying to determine whether this data set has been used by several different people to do different analyses and if so, which I understand it to have been, whether what we got turned over to us, to your knowledge, Dr. Nicholson, was all the different analyses that's available at Mount Sinai or whether it's just the computer analyses that would have been what you knew about on your disks?
MS. DIX: You are assuming you both understand what analyses you are referring to.
MR. SCHROEDER: Any analyses of these 2907 insulators.
Q. Do you understand my question,
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Doctor?
A. Well, the ambiguity is in the term analysis. I think an unambiguous analysis of the data has been done with respect to asbestosis by Steve Markowitz and others. That's published in the open literature.
Q. I understand. What --
A. And I don't know another one.
Q. Dr. Miller has done analyses of these people, hasn't he?
MS. DIX: Objection.
A. I don't know. I can't answer that.
Q. How about Dr. Ruth Lilis, she's done analysis of these 2907; is that right?
A. She certainly has been involved in what Steve Markowitz did. She's been involved in interpretation of the causes of death and that's -- but that's -- for example, mortality is nearing completion, but we have not done a mortality analysis, per se. We are in the process of working through one.
As I indicated, I don't know what Dr. Miller has in terms of analysis and I
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think I'm correct in other than Dr. Lilis' input to anything Miller might do or Markowitz might do, she has not, on her own, headed what I would call an independent analysis, but she's certainly been part of any analysis that's done in the past and may be ongoing

now.
Q. Did you turn over to Dr. Levin all statistical modeling and programs that you have created or run in connection with the 2907 insulators?
A. I don't have any such programs.
Q. Who at Mount Sinai would have those, do you know?
A. I don't know of such programs at Mount Sinai and here I am excluding programs that Markowitz and Miller might have, because I was not involved so much in that.
Q. Let's limit it to what you know about.
Based on what you know about of your own materials, have you turned all of that over dealing with --
A. Yes, I have, because I have not

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developed a program for the 29 -- the mortality analysis program for the 2907, that would be difficult to do so - but I do not have one.
Q. You said you were working on a mortality analysis of that same group presently?
A. Yes, working on a mortality study and when the death and information on them are as complete as I'm going to get them for the period of time, then I will conduct a mortality analysis comparing the numbers of death seen in this group to that which would be expected, having the noted exposure to asbestos.
Q. When will that study be completed?
A. Very shortly, over the summer.
Q. Is it in draft form already?
A. No, not at all.
Q. Is this a study that's being conducted under your direction?
A. Yes and I have the data available on that study as far as it has gone at this time.

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Q. Have you turned over all documents relating to any and all analysis of dose response relationships for an asbestos exposed individual?
A. I believe.
Q. Let me qualify that, for those who smoked.
A. I've turned over --
MS. DIX: Objection. Seems like there are analysis in existence.
Q. Are there analyses?
A. I've certainly done analysis and it's been published with respect to exposure of response relationships and they have discussed the smoking issue and what was

turned over was a criteria document I wrote for the EPA that was published in 1986.

I did risk assessments for the Department of Labor 1986 standard, which they used in the promulgation of that study and the enforced study. Those are - at least I can give you those.

I gave you what I had, which was I think the '94 standard and I produced the

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pages, the relevant pages.

Q. Are there other materials that relate to those analysis that you still have in your office?

A. Not that I know of, no. Not of the original papers that I would have doodled on or to put it together.

Q. Those are still around?

A. No.

Q. Doctor, item six on the subpoena was documents relating to analysis of smoking quit rates among asbestos exposed individuals; have you produced all of those?

MS. DIX: Again, objection to this. It assumes there are analysis in existence.

A. I haven't made such analyses. I discussed them with Dr. Harris and the data that would be utilized in such analyses are coming to you, you are getting the raw materials.

I have in this case looked at the documents that I produced for this case, looked at the differences of ex-smoking and

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smoking combined with asbestos exposure and what the combined risk would be and I did separately consider current smokers and ex-smokers and that's to my awareness.

My only analysis dealing with ex-smokers to the extent that it is a relative issue, it has been discussed in the EPA document, so what I might have done in the past would be there and I didn't do a quote/unquote analysis of ex-smoking as such.

Q. Not in the EPA or ever?

A. I think ever, but the issue would have - I hope it would be discussed in EPA documents.

Q. Just so that I'm clear, you said that you had analysis of smokers versus ex-smokers, to the extent you did one, you produced it to us; did I understand that right?

A. Yes.

Q. What document was that?

A. It's the August 30th document.

Q. So it's your expert report?

A. That's correct and it did look at

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the issue of smokers and ex-smokers and what the --
Q. I understand.
Before that report you had not done a separate analysis?
A. That is correct and I am just indicating I could have discussed it again in this EPA criteria document.
Q. Did your opinions in your expert report differ from the opinions in the EPA document?
MS. DIX: Objection.
A. I don't believe so. At that time it was certainly known that the smoking risk does decrease several years after cessation of smoking and thus the multiplicative interaction between asbestos and smoking would be less and that would be likely to have been discussed in the EPA document, but I can't guarantee you.
Q. What was the data in the EPA document?
A. I think it's June or July -- June 1986.
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Q. And that reduction of risk was well known in the literature by then; is that true?
A. Yes.
Q. Have you ever had any communications, Dr. Nicholson, with any tobacco company?
A. No. One might have sent me something, but it was asking for a blueprint, it was not a serious communication.
Q. So in connection with your work, you have never sought out any information from tobacco companies?
A. That's correct.
Q. Have you produced to us, Dr. Nicholson, copies of all correspondence and communications, if any, that you have had with unions in the United States dealing with the asbestos risk?
A. I have, but I don't have any. My correspondence goes in two weeks. I just don't keep it.
Q. Is it fair to say over the course of your career, you would have had fairly
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extensive correspondence with the unions?
A. No.
MS. DIX: Objection.
A. No, that's not fair. I would have had -- over the years I have had certainly occasional correspondence, somebody from some

union asks me about some asbestos questions.
During the 1969 to 1975 period of time, it would have been more, because I was - particularly with Johns Manville and the insulator's worker's union, keeping them apprised of what we were doing and so they would have a look at what the green sheets were going to be a bit ahead of time.
I would also have had communication with local union members in the setting up of examination sites in the 2907 survey, the '81 to '83, so we are - when we are doing something with the group then I would be involved.
Q. Were there others at Mount Sinai who would be responsible for having communications with the unions?
A. Not responsible, but certainly

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others at Mount Sinai would have had communications with unions and some far more than I did.
Q. Who would those people have been at Mount Sinai, I assume Dr. Selikoff himself?
A. Oh, yes. He was always receiving correspondence, so it's his prime place there, but depending upon this particular circumstance, Dr. Hyman, when he was there, Dr. Holiday, he is an industrial hygienist, would have, I'm sure, correspondence. I know Dr. Lilis does and I'm sure -- certainly Steve Levin and --
Q. And there was a point in time when Dr. Ritsey was on your staff?
A. Correct.
Q. He was a Johns Manville employee?
A. That's correct. That was part of the initial cooperation that was established. Not only were they providing some funding, they provided Mr. Ritsey.
Q. When was he part of your staff?
A. From 1969 through 1973 or '74, something in the early 70s.

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Q. Would it be fair to say, Doctor, there was extensive communication between Mount Sinai and Johns Manville Company during the 1969 to 1974 time period?
MS. DIX: Objection.
A. Yes, certainly through Mr. Ritsey. I mean he was totally informed of -- I mean of what we were doing, not only in the insulation industry program, but in others and I'm sure kept Manville well informed, which was absolutely appropriate.
Q. Doctor, rather than going through every document on this list, I may come back to some later, this is Exhibit 1, you looked at this list when you got it, did you not?

A. Yes.
Q. And to the best of your ability you responded to any documents that you had in your possession that related to this?
A. Yes, with the exception that I left 2907 material to be in the response at Mount Sinai, you know, I'm just --
Q. Okay.
A. -- emphasizing again that

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aspect.
MR. SCHROEDER: Mark this as Nicholson 2.
(Nicholson Exhibit 2, Risk Assessment, marked for identification.)
Q. Dr. Nicholson, I'm going to hand you what's been marked as Nicholson 2.
You recognize that as a document that came from your files, right?
(Handing.)
MS. DIX: Give him a chance to look at it for a second.
MR. SCHROEDER: Sure.
Q. It bears a production Bates stamp of WN with the number 6857, the WN being William Nicholson I believe?
A. I mean I may have made a slide of this at one point. I don't recognize the origin of this document, but it's familiar in context, but if I made it --
Q. What I want to ask you was - please hold onto it, sir.
I want to ask you, this appears to be a list of factors for a risk assessment; is

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that a fair statement?
A. Yes.
Q. And this is a list of factors you developed; is that right, sir?
MS. DIX: Objection.
A. I think it's a generally accepted risk of factors.
Q. You would endorse this list?
A. At a minimum, yes. There could be other factors playing a role as well, but these certainly all do.
Q. What I'm trying to get at since there is apparently some question as to whether you recall whether you drafted this or not, my point is you certainly agreed with what is on here?
A. Yes.
This apparently would be with respect to risk of disease from asbestos exposure.
Q. Yes.
A. And I certainly would agree with this.
Q. So if you want to estimate the

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risk of disease for asbestos exposure, these are factors you need to consider?

A. Yes.

Q. And are there factors that are not on this page that you would also need to consider?

MS. DIX: Objection.

A. You may wish, if there is the possibility of other exposures of consequence playing a role, you would want to know about that as well and you decide that after looking at individual histories or the consideration of the group in question. I'm just saying there could, right now I don't have something of equal consequence as these.

Q. Tell me in your view, Dr. Nicholson, what are the essential elements that you need to look at to assess risk for asbestos exposure in an individual?

A. All of these are.

Q. The first one listed is magnitude of exposure; please describe for me why you think that's important?

A. Because the degree of asbestos

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disease - disease or at least the major asbestos diseases, in particular lung cancer, mesothelioma and parenchymal fibrosis, depends on the amount of asbestos inhaled into the body and that's described on its intensity and the duration over where that -- over what that intensity occurred and here you are looking at averages in terms of when and you can obtain them.

There are some secondary measures in exposure that might come from body tissue analysis, but there is -- it's not as appropriate as knowing what the external exposure was at the time.

Q. Doctor, when you were -- you are no longer at Mount Sinai, you retired from Mount Sinai?

A. I'm retired, but I still go to work there everyday, the only difference is I don't get paid.

Q. A relationship they probably enjoy as well.

A. I hope so.

Q. Dr. Nicholson, at the time you

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were at Mount Sinai and you were dealing with asbestos issues from approximately 1969 up until today, did a large part of what you did at Mount Sinai involve the development of models and analysis of data relating from studies of insulators?

MS. DIX: Objection.
A. A fair amount, but it's --
Q. If there is a person at Mount Sinai who was the point person for that, would that have been you?
A. In terms of risk assessment, yes, but in terms of clinical information, the particular exposure circumstance would be someone else.
Q. Risk assessment would have been you?
A. In terms of mortality, yes.
Q. For asbestos-related disease?
A. Yes.
Q. We don't have on here frequency or is that included in one of these factors?
A. Well, it's included in the intensity and duration. The intensity is the

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average intensity over the duration and so you have the beginning exposure and end exposure and the best you can do about estimating the intensity over that duration, if you have circumstances where there is a period then a stop and a hiatus in the number of years, you want to do the same thing separately for the two different discreet periods of time.
Q. When you are looking at intensity, do you find that in some jobs asbestos exposed individuals can have very high intensity for very short periods of time?
MS. DIX: Objection.
A. Yes, that is certainly the case.
Q. When you are looking at intensity, you need to do that I take it on a job-by-job basis, that is intensity can be different for different jobs?
A. If a person is going to undertake different jobs over time, you would want to evaluate each of the jobs and so whether it be in a plant or in a field survey, for example insulators.
Q. For evaluating each job you would

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evaluate not only the intensity for that job, would you evaluate the duration as well for that particular job?
A. You would have to inquire of the worker how frequently he does that job and for how long and you get a measurement of the average exposure over a period of that job.
Q. If you were looking at an individual who may have asbestos-related disease, you would want to talk to the individual to find out what jobs they held and what they did within each job, is that fair?
A. In some circumstances where you are wanting to evaluate a single individual's exposure in absolute detail, in terms of a

study I do of a group of workers, you need to - you need not do that for each person, you do it of a group and consider the different jobs that an insulator does and evaluates them and weights the measured exposure according to the percentage of time on average that insulators do that job.

You can then assign two insulators a general average exposure obtained in this

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way.

Q. That would give you an average?

A. A very good measure of what the exposure insulators as a group had over time and certainly it is one that has changed, but we have done that.

Q. You testified in individual cases before, individual lawsuits?

MS. DIX: Objection.

A. Yes.

Q. And approximately 90 percent of your litigation work has been on behalf of plaintiffs, right?

A. More than 90 percent of them.

Q. In fact, you have had a long-standing relationship with Mr. Kazan here, haven't you?

A. Yes.

Q. And have you had the same with Mr. Motley's firm, Ness, Motley?

A. Very little there. I've also had extensive work with the Wortnick firm.

Q. That's the firm that Madelyn Chamber used to be a part of?

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A. She still is, I believe. I'm sure she is.

Q. She is now suing the tobacco companies, isn't she?

A. I don't know.

Q. In the individual cases where you have testified, you testified that you would need ventilation information to determine the risk for any individual; isn't that correct?

MS. DIX: Objection.

A. You may not need ventilation information, but you would need some information on exposure to calculate a numerical risk at points in time and that is in many individual cases not information that is readily available.

Q. But you have testified in individual cases you would want ventilation data, isn't that right?

MS. DIX: Objection.

A. If I needed to know an exact exposure for a particular job, you would want to have as much information about what that exposure would be.

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I mean I need -- I don't need ventilation information if I had a very good measure of a person's exposure by just damaging data in a particular facility.
The actual amount of information needed on exposure varies with the particular case.
If you have an individual that's had exposure as an insulator or factory worker, something like that, over time A to B and he has mesothelioma and he has had one exposure to asbestos, I'm quite certain that that one exposure to asbestos would be the cause of this mesothelioma and would so testify.
Without knowing what is the description of it, he talked about working as an insulator for five years back in the past, and has had no other exposure that he knows of and I have no information that he did have another exposure, then that exposure is it and I don't need a measure.
Q. If somebody has held more than one job, let's say two or three different jobs
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over the course of several different years, in those circumstances, haven't you in the past taken the view that you would want to have information on the ventilation, that is the air fibers contained in the work area where the individual worked, in order to make a risk assessment?
A. It would be desirable, indeed, and I understand now what you are saying about ventilation.
Ventilation per say could mean what's the airflow coming into the room; what's more important is the air exposure to the individual.
Q. Maybe we were talking past each other.
A. It's all right, let me answer the question that you asked.
Q. Okay.
A. To evaluate the contribution of job A, versus job B, versus job C, you certainly want time factors to be present, because they do count and you would want as much information on exposure as possible.
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Sometimes you can very clearly say that exposure A is much greater than exposure B.
For example, a five-year insulator is quite different than five years as an automobile mechanic, so there is a background

of information that can be used, but in many circumstances you don't have that and there you can't say exactly this -- I know this job has more exposure than that job, you can say they may be qualitatively similar, but I can't say one versus the other is the greater.

You do the best you can. In many cases you can't do much more than make a qualitative statement.

Q. With respect to exposure, you have taken the view in the past that there is no safe level of asbestos exposure?

A. Yes and I still take that view. The data we have do not indicate the existence of a threshold and so the simplest model that one uses is a linear threshold model, but certainly at very low exposures and at very low risk.

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Q. Have you been critical of the OSHA standard that's in effect for permissible levels of asbestos?

MS. DIX: Objection, which OSHA standard?

Q. The present OSHA standard.

A. Well, it certainly carries a risk and I stated what that is and they have themselves in these publications, so it's recognized by OSHA and by everyone.

Q. What is the present OSHA standard for permissible levels of asbestos?

A. 0.1 fibers per milliliter on an eight hour time waited basis.

Q. Even at that level, OSHA's position is that level presents a grave danger to a worker, right?

A. I'm not sure.

MS. DIX: Objection.

A. Of their terms, but it certainly presents a danger. Risk for a 40 year employment is three deaths per thousand or more than three deaths per thousand and OSHA, I know, is concerned very much if the risk is

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even one in a thousand and the difficulties of measurement are the reason that OSHA has not produced a lower standard, but for other reasons. Exposure to workers in the United States has decreased very, very dramatically and for which we can be thankful.

Q. Even short-term exposures to asbestos causes elevated risks?

A. I believe they do.

Q. It's your view - I think I understand from your prior testimony that the carcinogenic process can be associated with one fiber interaction; is that correct?

A. I don't know if it is or not. Theoretically it could be. You can imagine,

but we don't have biological data that it does, but in principal you can imagine that a single fiber scrambling to the NA in some way that leads to a malignant transformation, that's the cell divides and so it goes.

Simply put, it doesn't matter too much, because we know there are cases of asbestos disease developing at very, very low exposure, such as that occurring in someone

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living a mile from an asbestos factory, from the emissions of that.

We know it's occurring at levels certainly below the OSHA standard and whether it's exactly one fiber or not, efforts for controlling -- the rational for controlling asbestos to its low level as possible with the assumption that there is no threshold is absolutely clear.

Q. And that's reflected by the danger of even low exposures?

A. Yes.

Q. In considering risk assessment, fiber types are important I take it?

MS. DIX: Objection.

A. They play a role, but a relatively minor role, remarkable.

Q. Is the risk of carcinogenicity different for different fibers types?

A. It's very similar for chrysotile, amosite and crocidolite for lung cancer and tremolyte also. For mesothelioma the risk is similar for amosite, chrysotile and maybe two to four times greater for crocidolite.

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Q. For lung cancer, what is the magnitude difference between the risk for the different fibers?

A. Very little.

Q. What do you mean by that?

A. The uncertainty in the process of measuring a risk in a particular environment is greater than the difference across average value use for risks found for the amosite, chrysotile and crocidolite.

In other words, our best measure is that the risk is similar for lung cancer across all varieties of asbestos, but there is a factor of too uncertainty about that statement, certainly.

Q. A factor of two uncertainty?

A. Yes.

Q. Okay, what do you mean by that?

A. Well, we have very limited data on crocidolite. That which we have indicates a lung cancer risk similar to chrysotile and amosite and in the case of risk of chrysotile, you can find a factor of two different from a study of a textile mill in North Carolina, in

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a different factory producing chrysotile products elsewhere.
Q. When you say factor two, you mean twice as much difference?
A. Yes, so there can be -- the risk in some circumstances is roughly one percent per fiber year. In a textile plant in South Carolina it's about two percent per fiber year, but there is an uncertainty of that because of the small numbers involved and so in particular processes, you have that uncertainty present, but overall, the best estimate is that which I just gave you that on average the risk as we know it now appears to be similar with respect to lung cancer for the different - the major asbestos fibers.
Q. Just so that I understand, in your opinion, the risk for lung cancer is what you call similar with the caveat it could be different by a factor of two?
A. On an individual.
Q. Individual-by-individual basis?
A. An individual location-by-location basis.
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Q. The next thing you had on the Nicholson 2 Exhibit is age; why is age important?
A. Because if you get an exposure at later age you have less time over the rest of your life for the disease to develop and in the case of mesothelioma particularly, the risk rises expedientially from the time of exposure, so exposure at age 50, the lifetime risk is much less than a similar exposure at age 20.
Q. Because it's a time dependent relationship?
A. That's correct and it is also for lung cancer. Lung cancer risk is one that increases with time from exposure also, because what asbestos does for lung cancer is multiply the underlying risks of lung cancer and that's when it is rising with age and so...
Q. So our risk for lung cancer, as individuals, rises with age naturally?
A. Yes.
Q. And asbestos multiplies that risk?
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A. Yes.
Q. Time from exposure is your next element.
A. The same - for the same reason.
Q. Is that what you call latency?
A. Well, there is latency in that.

Q. This is time from first onset, is that what you mean by time from exposure?
A. Yes, time from exposure. We've already defined exposure in terms of duration, so you can deal with that. It also comes in the time analysis.
Let's just consider a short exposure so we don't worry about the duration aspect. Again, there is - for lung cancer, there a latency of about ten years before a risk is manifested, then it's one that multiplies the underlying risks and again as one gets older and the time from exposure increases, the risk increases and with mesothelioma, even without dealing with the age process of cancer, it's one in which the risk is greatly increasing with time from onset of exposure and thus one has to put in

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that factor if you are going to make a calculation of the risks of a group or an individual at some point in time when you have the appropriate information.
Q. So time from exposure is really two components, time from first exposure and the latency from --
A. Well, latency is in there and duration of exposure is in there too. They all have to be put in, but the time from exposure plays a role, that is you have a risk formula for either mesothelioma or lung cancer that has all of these factors in it in the equation.
Q. Are all of these factors that we have talked about so far equally important or are some more important than others?
MS. DIX: Objection. Equally important in terms of what?
MR. SCHROEDER: Themselves.
A. It depends upon the circumstances. In some cases the magnitude is what counts and other cases it's the time that counts.

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Q. Time from exposure?
A. That's right. If you are looking at a --
Q. Would it be fair to say, Doctor, if somebody had a brief, but intent exposure, that would be important and if somebody had a lower, but longer duration exposure, that too would be important?
A. That's correct.
Q. Is that what you mean by it depends?
A. That's right, it depends on what you have and where you are looking. If you are looking at something short, obviously the time from exposure may not be as important as

the magnitude.
Basically they all -- particularly the magnitude of exposure and the time from exposure are equally important. The age may not be important if you are looking at circumstances, you know, from age 20 to 50 or something like that and - but again, it depends on the group. That's all I can say.
Q. All right.

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Then you have listed other factors and you have plural, then you have listed after that cigarette smoking.
MS. DIX: I am a sorry, plural, sorry.
A. One of the other factors certainly is cigarette smoking.
Q. What other factors in addition to cigarette smoking; you mentioned one before, I don't recall what you said?
A. Could be exposures to other carcinogens.
Q. What other -- well, that's for purposes of, for example, a lung cancer analysis, because lung cancer is multifactorial?
MS. DIX: Objection.
A. Well, it need not be. You can get lung cancer from exposure to asbestos alone. You can get lung cancer from exposure to cigarette smoking alone, in terms of major external factors.
Q. Or you can get it from beryllium?
A. Yes.

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Q. Or from --
A. A host of other agents.
Q. And when you are looking at a group that's occupationally exposed, those other agents are potentially causative as well?
A. You have to consider them if they are present.
Q. If you were looking at an individual in a lawsuit, you would want to know those factors as well for that individual's exposure, would you not?
MS. DIX: Objection.
A. In an individual lawsuit, yes. You can - if you have a group, you can make estimates of whether they might play - other factors might play a role or not.
Q. Certainly you are not saying that when looking at a group of people you do not need to look at other potential occupational exposures for cancer?
A. You don't have to consider it for every person. For example, with insulators, you have these guys going to work around the

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age of 20; possibility of an exposure to something before exists. The average insulator, his exposure to asbestos is going to be very dominant.

There could be peripheral exposures occasionally to something else in a plant they might work in, but on average for a large group of insulators, at least from what - at least the insulators we have worked with, their exposure of concern is to asbestos.

You have in some unique circumstance people hired by some chemical company working in a particular chemical plant that produces agent X that is very carcinogenic and obviously you would take that into account because you know it's there.

Q. Let me make sure I follow that.

For purposes of determining the risk of lung cancer to an asbestos exposed population and that is the risk from asbestos, is it your testimony that the asbestos, for example, in insulators is generally so overwhelming that the other carcinogens that

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they may be exposed to become less important?

MS. DIX: Mischaracterizes prior testimony.

A. For the case of insulators as a large group with multi circumstances, insulators across the United States for example, the likelihood of other exposures of consequence is very low and so the data you get on, for example, in the 17,800 insulators who are working across the country is very directly applicable to an asbestos risk.

I noted that there could be localized circumstances where you would, for a localized group, want to consider other exposure, but for this group from all our experiences over at Sinai, now since the middle 1960s, it is very clear that other than cigarette smoking, other exposures that might occur in various circumstances in their work play a minimal role in their disease.

Q. If you had available to you information of known other exposures, would you want to take that into account?

A. Yes, I said if you had it, you

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would use it in the particular circumstances where it is important, but on average for that group, the analysis that Selikoff has done is very appropriate to asbestos exposure and cigarette smoking.

Q. Dr. Nicholson, you testified in a

case called Louis versus Ray Bestos, Manhattan?
A. Yes.
Q. Do you recall that?
A. I recall having testified at it, I don't recall any details of it at this point.
Q. In that case you testified that in most circumstances you cannot determine exposure because of lack of information about exposure?
MS. DIX: Are you asking if he recalls that?
Q. Do you agree with that?
A. I found that to be the case in many individual legal circumstances and we've gone through that already here where it's clear in the past that I have been confronted with one individual having two or three

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exposures and being unable to say that one is greater than the other.
I can say that maybe these two are certainly greater than the third, but I can't rate one versus the other, so you have asbestos guy explains his exposure you still have -- there is still uncertainties about it.
Q. In those cases you were unable to draw a quantitative conclusion; is that correct?
MS. DIX: Objection to the form of the question.
A. That would be correct, I could not say what the exact exposure was to this person.
Q. Would you agree with me, Dr. Nicholson, that a job title for a person exposed to asbestos would be more accurate than an occupation title or an industry title for measuring exposure?
MS. DIX: Do you understand what he is --
THE WITNESS: I understand.
A. I think in general that is the

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case. There are again exceptions and I go to the insulators.
Being an insulator is more descriptive than a job that he might have done here and there, because they do so many different jobs. The standard insulator has a lot of jobs.
Q. Apart from the insulators?
A. In general that would be the case and even with insulators, if you come across someone that spends his whole life in a fab shop which is different than the insulators and their job plays a more important role than the insulators, so across many jobs it can go either way, but I think in general a job may

be more useful. Certainly it is - sometimes in other cases the industry is adequate.

Q. Dr. Nicholson, would you agree with me that one of the difficulties over the years in measuring asbestos -- risk for asbestos disease, I should say, has been getting an accurate measurement of actual asbestos exposure for the individuals?

A. That certainly is a case for many

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individuals, others we can do fairly well. I mean being told that someone was employed in the 30s in an asbestos textile mill tells me a lot about that exposure.

Q. You excluded --

MS. DIX: You have to make sure -- are you finished?

A. Again with the insulators we have good data over the years, so a job history there in terms of when he worked provides good information, I believe, on his cumulative exposure.

Q. The textile mills, you excluded those from consideration in your expert report, did you not, when you were discussing lung cancer, do you recall that?

A. I think I included in the report references to textile work. I think or I know I have got a couple of articles by Barry and I think one of them involved textile -- at least one of them involved a textile mill.

I have to look, but I believe there is somewhere in the bibliography and in the - not directly in discussions, but

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information --

Q. Would it be fair to say, that putting the insulator's studies aside, in the balance of the studies of asbestos exposure getting accurate exposure measurement has been difficult?

MS. DIX: Objection to the form of the question.

A. Well, we've got a lot of jobs where we have very good -- very good information that can be used. Shipyard workers for one, brake mechanics for another.

Q. Doctor, my question was and it was designed to elicit a yes or no, you are entitled to explain. The question was whether or not you agree, generally, apart from the insulator's studies getting accurate measures of exposure has been a challenge?

MS. DIX: Objection.

A. I'm saying no because we have information in many large and important trades what the average exposure might be for a group of workers, not for an individual in one of these, because -- but to conduct a study of a

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large group of workers, one can assign a very reasonable exposure in at least the circumstances I gave.
Q. Would you say it's a fair statement to say that if you have a cohort of people exposed to asbestos, that those - and let's take the disease of lung cancer, those who eventually present with a disease of lung cancer are more likely those in the cohort who had the heavier exposure to asbestos?
MS. DIX: Objection to the form of the question.
A. It if you had a cohort with largely varying exposures, the likelihood of a person with lung cancer coming from -- if you take into account the numbers in each of the groups, the possibility of a person in the higher group developing lung cancer is greater than the lower exposed group.
Q. And so if you take a look at a cohort of a thousand people exposed to asbestos in an occupation and you are checking them for lung cancer and out of that group a
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hundred present with lung cancer, would it be fair to say that those hundred who presented with lung cancer are among the more heavily expose in that cohort?
MS. DIX: Objection to the form of the question. Are you presenting a hypothetical to Dr. Nicholson? I'm not following the question.
MR. SCHROEDER: I will stand by my question.
A. It depends on the group first and when the people came to work, even in a group which all jobs had equal exposure and you take a group of people starting employment at age 20, it's likely that there is a greater likelihood to develop lung cancer the longer one is exposed in that group, so you have that.
Q. Let's assume everything else being equal in the group, that is they all started the same time, okay and that they were all the same age, then my question is if that's a group of a thousand people within a trade and within a certain latency period a hundred of
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them develop lung cancer --
A. And they all smoked the same?
Q. And yes, smoking habits are not different among the group, yes, let's assume that smoking habits were all the same, because the risk for lung cancer is a dose response

relationship to asbestos, isn't it fair to say the hundred people who got lung cancer are most likely among the more heavily exposed to asbestos within the group?

MS. DIX: Objection to the form of the question.

A. Given there existed quite an adversity of exposures, it is likely that the chances of having developed a lung cancer is greater for those in the higher group than in the lower group.

The chances -- the question you are asking depends very much on the population in those groups. I'm trying to get that out of it, because if you have a tiny group heavily exposed and a large group lightly exposed, you are going to have more cancers from the --

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Q. Right.

MS. DIX: Can you let him finish?

A. -- from the lower group, so I'm trying to answer your question in the positive that the higher exposed group will have the greater risk of developing lung cancer and to the extent that you have equal population of exposures, there will be more coming from that group than from the other people -- size group.

Q. Let me make sure I follow that.

If we have a thousand people in our group and everything else being equal including smoking habits, a hundred of them present 40 years later with lung cancer, those hundred people are more likely to have been among the more -- more heavily exposed to asbestos than as a group - the other 900 in the thousand, fair statement?

MS. DIX: I have a continuing objection to this line of questioning.

A. If you consider just heavily exposed and lighter exposed, two groups --

Q. No, I don't want to do that.

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What I want to do is --

A. The answer is not necessarily, that's what I'm trying --

Q. What would it depend on?

A. Upon the size of the heavily exposed group compared to the other group. If you have a tiny number -- you have ten people in a heavily exposed group and a hundred people in a group exposed to half as much, you are going to get a lot more in the lesser exposed group, so if you have -- that's what I was going to say.

Try a heavy exposed group and a light in equal size, you will get more from the heavily exposed.

Q. If they are all mixed up in a group of a thousand, the heavily exposed and lightly exposed, if you get a hundred that eventually present with lung cancer, is it your testimony that they would more likely be coming from the heavier exposed group?
A. If the two groups are equal in size, yes, with the other things being equal.
Q. And that would be a reflection of

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the dose response relationship for lung cancer and asbestos?
A. Yes.
MS. DIX: Do you want to take a break.
MR. SCHROEDER: That's fine.
THE VIDEOGRAPHER: Going off the record it's 11:39 a.m.
(Recess taken.)
THE VIDEOGRAPHER: We are back on the record continuing the deposition of Dr. Nicholson, it's 11:46 a.m.
MR. SCHROEDER: Thank you.
Q. Dr. Nicholson, let me switch to a little bit different topic if we can.
If you have an asbestos manufacturer who's been sued in a lawsuit with 49 other asbestos manufacturers by an individual who's claiming personal injury, I want you to assume that for a minute, okay, so you got a plaintiff suing 50 asbestos companies, okay and one of the 50 companies says that that's not my product, because it's really the product of the other 49 asbestos

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companies, do you know of any way that that one asbestos company can accurately determine the amount of exposure to their own product?
Apart from the label on the name of the product, is there any way to look at the individual and determine the amount of exposure that an individual had to a particular asbestos manufacturer's product?
MS. DIX: Objection to the form of the question.
A. You are speaking of 50 manufacturers of the same product?
Q. Let's do it this way. Let's assume you have an individual.
A. The individual would know what products he worked with.
Q. Apart from the names, you have an individual and the individual is -- all you know is the individual is exposed to asbestos, okay and all you know is that at some point in time the individual says that they were exposed to the 50 different manufacturer's products.
I want you to assume that for a

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minute, okay, but understand that, the individual can't recall what the time periods where that they were exposed to any particular product, okay, you follow me so far?
A. All right.
Q. Do you know of any way for one manufacturer to determine the amount that an individual might have been exposed to that manufacturer's product?
MS. DIX: Objection to the form of the question.
A. Not directly, unless there was some circumstance where the work area this person was in was one that was - in which it was impossible for the manufacturer to have ever had a product.
I mean you can have something like that that would be helpful, but in terms of tissue analysis or something like that, that would not -- considering similarities of products, that would not be practical.
Q. I want you to assume one other scenario now.
You have the same 50 manufacturers
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and manufacturer number one -- I'm sorry, same 50 manufacturers and the plaintiff recovers a verdict against all 50, okay, you follow me?
A. Yes.
Q. You need to respond audibly, I'm sorry.
A. I'm sorry.
Q. Plaintiff has a verdict against the 50 and now one of the manufacturers wants to recover from the other 49, because the manufacturer says that it was mostly somebody else's product.
Same question, do you know of any way to tell accurately the amount of that one manufacturer's product that the plaintiff may have been exposed to, versus the other 49?
MS. DIX: Continuing objection to the form of the question.
A. Absent direct information on the person working product by product, no.
Q. Does the absolute risk of asbestosis-related disease increase with time?
A. Yes, but for a given exposure.
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Q. I want to talk about asbestosis if we can.
The relationship between asbestos and asbestosis is not linear, is it?
A. That's correct.
Q. Presently, the data are

insufficient to establish a dose response model for asbestos and asbestosis, isn't that correct?

MS. DIX: Objection to the form of the question.

A. We have some information on dose response models, but it is limited.

Q. You recall the case of Ottwater versus Owens-Corning which you testified on in July 1997?

A. No.

Q. I'm going to show a copy of your deposition in that case and see if that refreshes your recollection.

(Handing.)

A. I don't remember saying those words, but it's roughly what I'm saying now.

We don't have a -- we don't have

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an ideal dose response model, it depends on the various factors indicated there.

Q. In this case, you testified, did you not, that you said, "I just would emphasize that a model for the exposure response relationship for any measure of asbestosis, whether you are talking about pleural plaques or a one slash zero reading or whatever, you have a model that is not linear. It depends on intensity and duration of exposure. It depends on the time from first exposure to when it is read. It is a model that's even now quite uncertain and is especially so at the lower ranges," right?

A. Yes.

Q. And do you agree with that testimony today?

A. I might soften the statement as to the uncertainty. I think I would say -- I would not say it as harshly as I did there.

Q. It is still uncertain, is it not?

A. Yes.

Q. What has changed between your deposition on July 30, 1997 and today that

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would affect your statement?

A. I think probably more study on the issue on my part.

Q. Following that study, it remains uncertain?

A. Yes. What we don't have is -- we have good information on risk versus exposure at a particular point in time, we don't have good information on how that risk will increase with time and at what -- and at each of the different levels, so we know it does and we -- but we are dealing with a three dimensional graph.

Q. Are you still studying the issue?

MS. DIX: Objection.

A. Not at the moment.
Q. You testified in the Fiber Board Asbestos bankruptcy, did you not?
A. Yes.
MR. KAZAN: No, you didn't. It is not in bankruptcy.
Q. Did you testify --
A. No, then.
Q. Did you testify in Selia

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(phonetic) Texas bankruptcy?
A. Not to my recollection.
Q. Did you testify in hearings for Fiber Board?
MR. KAZAN: Are you talking about the Fiber Board class action?
MR. SCHROEDER: I'm going to find out here.
MR. KAZAN: If we are in bankruptcy I better get back to my office.
MR. SCHROEDER: I don't mean by that question to impugn anybody's business interest.
Q. Did you testify in hearings relating to estimation procedures for Fiber Board?
A. What type of estimations?
Q. For future claims.
A. I may have. I certainly was involved with that process for Fiber Board.
Q. In connection with that process which occurred when?
A. Several years ago.

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Q. In connection with that process, you took the position at that time that there was no sufficient dose response relationship that could be established for asbestos and asbestosis, right?
MS. DIX: Objection to the form of the question. Are you asking whether he recalls that prior testimony?
MR. SCHROEDER: No, I'm asking whether he made that testimony.
A. I don't remember what I made at all.
Q. You certainly have taken that position in the past, right?
MS. DIX: Objection.
A. I've taken the position that we certainly cannot make estimates of future disease to the accuracy that we can with mesothelioma and lung cancer.
One certainly can say that given some exposure circumstances, it is clear that there will be some degree of asbestosis present in a particular group.
One can make qualitative estimates

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of asbestosis risk and that was possible at that time, it isn't as far from accurate as for the malignancy.

Q. You know Dr. Francine Rubinowitz, do you not?

A. Yes.

Q. You have advised Dr. Francine Rubinowitz in the past, have you not?

MS. DIX: Objection to the form of the question. What do mean by that?

A. I haven't --

Q. You have advised Dr. Rubinowitz in the past that the data are presently insufficient to be able to perform a dose response model with certainty for asbestos and asbestosis; isn't that right?

MS. DIX: Objection to the form of the question.

A. I don't remember doing so with Dr. Rubinowitz. I certainly had conversations and may have said something equivalent to that, that you cannot make an estimate with anywhere near the certainty that you can for lung cancer and mesothelioma.

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Q. Just to be sure now, Doctor, you qualified that because you said not with anywhere near the certainty as you could with lung cancer and mesothelioma.

What I want to ask you is presently, as we sit here today, you can't develop a dose response model for asbestosis from asbestos exposure with a statistically reliable degree of certainty, can you?

MS. DIX: Objection.

A. I don't know what you mean by statistically reliable degree of certainty, that's a totally arbitrary term. You can develop a dose response model that can have some uncertainty associated with it.

I think the dose response model is more inaccurate for nonmalignant disease than for malignant disease.

Q. At present because of that inaccuracy, you don't have a dose response model from the data that you can present for asbestosis, do you?

MS. DIX: Objection.

A. I don't, nor was I asked to do

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one. I might have produced something if I looked at it, but it's a topic that I have spent limited time with.

Q. You certainly spent enough time up to 1997 when you made the statement in the Ottwater case, did you not?

MS. DIX: Objection.
A. Spent that much time with a nonmalignant disease up to then, that was not a major aspect of my work at all.
In the statement I said and I would stand by, except I would modify it a bit as I indicated, because I have spent more time since then on the issue of nonmalignant disease.
Q. So to rounds out this line of questioning, you stand by the statement -- I'm asking you this: Do you stand by the statement in the Ottwater case modifying it now to say that it's not quite uncertain, but it remains uncertain; is that correct?
(Handing.)
A. Let me look at that.
Yes, I would take --

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Q. Out the word quite?
A. Quite.
Q. So that the record is clear, then, it would read it is a model that even now is uncertain and is especially so at lower ranges, correct?
A. Yes, I would say that does not preclude a model from being created though.
Q. And you have not developed one?
A. That's correct.
Q. What's your definition of asbestosis?
A. I don't have one explicitly. Certainly asbestosis at some degree of parenchymal fibrosis, one could arbitrarily consider pathologically observable fibrosis asbestosis or one might require there be sufficient scarring to be manifest on X-ray.
It's not a term that I really worry about, because if I used it, I would characterize what the degree of abnormality was.
Q. Your degree of abnormality would be characterize by an ILO rating?

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A. Yes.
Q. What level of ILO rating do you use for abnormality?
A. In utilizing data of others, I would use in general one slash not, which indicates that the reader believed there to be an abnormality present, but it depends upon the circumstances, sometimes if the data detailed you would use one slash one.
Q. A one slash zero rating means not only that the reader says that -- the reader finds evidence of abnormality, but that other readers might not find the evidence, isn't that a fair characterization?
A. I think it's more that he gave

serious consideration to a possible zero; in other words a zero slash one. I'm sure that at any -- even at higher readings, some reader could consider that, another reader would be vastly different, either way.

Q. It would become important at the one over zero range, would it not, because zero is evidence of no disease -- I'm sorry, zero would be no evidence of disease and a one

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would be evidence of disease, right?

MS. DIX: Objection. Tom, what is your question?

Q. The question is you said that other readers could differ, but my point is it makes a difference at the one over zero level, does it not, because that's the cutoff for whether there may be evidence of disease, that is a one versus a zero?

MS. DIX: Objection.

A. And that ambiguity is why there are lawyers arguing about it.

Q. It's not just the lawyers, actually the American Thoracic Society takes the position that one over zero is not evidence of parenchymal abnormality, isn't that correct?

A. I don't know.

Q. Would you agree with me, Dr. Nicholson that a one over zero rating under the ILO is not a clinical diagnosis of asbestosis?

MS. DIX: Objection.

A. I earlier said that that could be

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the case. The term asbestosis is an ambiguous one. Some people could utilize it at that level, others would utilize it at a higher level.

It is even possible that one could utilize it at a pathological level when you have analysis of a lung tissue specimen showing scarring in an individual that has a normal X-ray, so I'm not one to say what quote/unquote asbestosis -- how asbestosis should be defined.

Q. You are not a medical doctor?

A. That's correct.

Q. The studies that look at prevalence of asbestosis among smokers are studies that, as reported in your report, are studies that almost universally use as a measure of potential asbestosis an ILO rating, right?

A. Yes.

Q. And that ILO rating is almost universally, in those studies, a cutoff of one over zero?

A. That's correct and I stated that

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and I gave the data that were available and where the cutoff was different. I used what was there and it was sufficient to indicate there was a relationship between asbestosis exposure and smoking.
MO MR. SCHROEDER: Move to strike.
Q. My question is simply that a one over zero ILO rating of a parenchymal abnormality is not the equivalent of a clinical diagnosis of asbestosis, is it?
MS. DIX: Objection.
Dr. Nicholson stated he was not a medical doctor.
MR. SCHROEDER: He issued an opinion in the case and I want to get to the bottom of what that opinion was.
A. I'm not using the term asbestosis.
Q. Okay, so would you agree with me a one over zero ILO rating by itself is not a clinical diagnosis of asbestosis?
MS. DIX: Asked and answered, objection.
A. It depends how one defines the term and I'm not defining the term, so I can't
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answer that question.
I will say one slash not is generally a measure of the presence of parenchymal fibrosis and depending upon the reader - in some it's virtual certainty of presence and others it may not, but it is strongly, so on average --
Q. You are aware that the American Thoracic Society set standards for the diagnosis of asbestosis?
A. Yes.
Q. And you have been aware of that when you have conducted these various studies you have of parenchymal fibrosis, right?
A. I haven't conducted such studies; I don't read X-rays.
Q. You collect the data?
MS. DIX: Objection.
A. I will utilize some of the data that is collected, but I have done relatively limited work with asbestosis, with clinical asbestosis.
I certainly get involved -- I certainly have been involved with mortality
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from asbestosis where one isn't arguing about what one slash not means.
Q. Are you, Dr. Nicholson, in your report when you say that there is evidence of a 50 percent increase of parenchymal abnormalities among smokers, that's your

opinion, right?
A. That's my opinion. It was clearly shown by the data of a dozen or more studies.
Q. That's your opinion, okay. When you offer that opinion, do you intend for that to mean that there is a 50 percent increase in clinical asbestosis among those people?
MS. DIX: Objection to the form of the question.
A. I take it to be exactly what was presented in the papers that were utilized. They were indicated that some were one slash not and some were one slash one and it's clearly an evidence of an increase across those exposure categories and by virtue of seeing an increase, it's clear they are looking at something that is not normal.
Q. And so my question was are you

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equating that with clinical asbestosis?
A. I have said I am not defining clinical asbestosis, I'm keeping out of that whole issue for the moment.
MS. DIX: If you have a particular part in the report, it might help to show Dr. Nicholson.
Q. So your opinion is staying out of the debate of what is asbestosis, is that your testimony?
MS. DIX: Objection, mischaracterize his prior testimony.
A. At this point in time I'm not wanting to make a definitive definition of asbestosis. It's not something I have considered directly, nor reviewed extensively in terms of the use of that term.
MS. DIX: His terminology is parenchymal abnormalities. I don't know what point, if any --
MR. SCHROEDER: I'm trying to get to the bottom of this.
A. I don't see what my knowledge or lack of knowledge of the explicitness of

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asbestosis --
Q. Well, you understand in a court of law, because you have testified in cases, that if somebody's suing an asbestos company, they are suing for -- if it's fibrosis, they are suing for asbestosis, right?
MS. DIX: Objection to the form of the question.
A. Well, they are suing for disablement.
Q. But they are suing for an asbestos-related disease, first of all?
A. Yes.
Q. And asbestosis is caused by asbestos, right?

A. Yes.
Q. It's not caused by smoking?
A. That's correct, by definition, but smoking can certainly play a role in the degree of asbestosis among people exposed to asbestos.
Q. That's my question, because by making that statement you have now equated the parenchymal abnormality studies to a clinical

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definition of asbestosis, which you told me you weren't going to do?
MS. DIX: Objection.
Q. My question is in your report you talk about parenchymal abnormalities, correct?
A. Yes.
Q. You don't talk about clinical definition of asbestosis, do you?
A. I don't believe so.
Q. And --
A. It may have been inadvertently -- I may have inadvertently done so, I don't recall.
Q. If you did, did you mean to?
MS. DIX: Objection to the form of the question.
A. I did not - I would not have meant to do so, because I was dealing with what people were observing, parenchymal fibrosis and as I indicated, I'm unclear as to the exact terminology of what everyone or anyone would use for asbestosis, so...
Q. Since you are unclear of the clinical diagnostic definition of asbestosis,

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is it fair to say, Dr. Nicholson, that you cannot give an opinion as to whether smoking increases the prevalence of clinical asbestosis?
MS. DIX: Objection, mischaracterizes his testimony.
A. Yes, I can, because I consider parenchymal fibrosis across categories going up to one slash two or -- I don't know, something involving a very high level that I'm sure anyone would call asbestosis and at that level cigarette smoking is doubling the risk, is doing so across degrees - of all degrees of abnormality.
Q. Would you agree that the ILO rating is one of the diagnostic tools for a diagnosis of asbestosis?
A. Yes.
Q. Would you agree it's not the only tool?
A. That may be the case. I don't have the explicit definition. I don't know if you need pulmonary function deficits or not, so that term, as I find that term used, it is

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very ambiguous, that's why I -- used by others at very low levels of diseases manifest clinically and it's certainly at higher levels.

Q. Would you agree that as time passes any effect that you may report on smoking on asbestosis is reduced?

MS. DIX: Objection. Tom, Dr. Nicholson is repeatedly saying -- he is using the term parenchymal abnormalities. I object.

MR. SCHROEDER: He has said it backwards and forwards, every which way. State your objection.

MS. DIX: If you have an issue point him to the part of his report. The transcript is getting inherently unclear here.

MR. SCHROEDER: Let's not leave the room without being clear. I am not leaving until it's clear and I agree with you.

MS. DIX: Do you want to know Dr. Nicholson's --

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MR. SCHROEDER: It's all of the studies in his report.

MS. DIX: What is your question, Tom?

Q. My question was just now, would you agree and I will substitute the word parenchymal abnormalities, that as time goes on any purported effect smoking might have on parenchymal abnormalities is reduced?

A. No, I wouldn't agree with that. Smoking is continued, there will continue to be manifest the same increase in parenchymal abnormalities in smokers compared to nonsmokers.

Q. Do you disagree with the data that Dr. Lilis found in her 1986 study that by the time you are 40 years since first onset, there is no apparent smoking affect on parenchymal abnormality?

MS. DIX: Objection to the form of the question.

A. At that particular study, it wasn't evident at the lowest level of abnormality. I don't know whether -- I think

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it was one slash not, because over 80 percent of nonsmokers had parenchymal abnormalities, so you are looking at something at the top of the range.

When you go on that same study and look at the two level abnormalities, there is

a twofold effect in that group -- at that level.

MR. SCHROEDER: Let's change tapes.

THE VIDEOGRAPHER: Going off the record it's 12:18 p.m. This is the end of tape number one.

(Recess taken.)

THE VIDEOGRAPHER: Back on the record continuing the deposition of Dr. Nicholson. This is the beginning of tape number two. It's 12:19 p.m.

MR. SCHROEDER: Let's mark this as Nicholson 3.

(Nicholson Exhibit 3, Graph, marked for identification.)

Q. I'm going to hand you Exhibit 3 to your deposition.

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(Handing.)

Do you recognize that as figures one and two from your report in this case?

A. Yes.

MS. DIX: This is the August 30, 1999?

MR. SCHROEDER: I think he only issued one, it's the September 1999 report.

MR. KAZAN: If you look at the first page of his report, his report is actually dated August 30th on its face, although it was submitted on September 1.

MR. SCHROEDER: Okay, but there is only one, right?

MS. DIX: Correct.

Q. We will call this your report, okay?

A. I'm not sure this is, because data have been erased.

Q. What data?

A. The two slash one smoking slash nonsmoking ratio. You have a label there, you

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don't have the data down there.

Q. Where?

A. Down here, that's what is missing.

Q. Do you want to compare it to a copy of your report?

This is what was produced to me.

MR. SCHROEDER: Is it the same.

MS. DIX: I think it's just a better copy. Do you have a copy of the report with you?

MR. SCHROEDER: I have my copy.

MR. KAZAN: These are photocopies.

MR. SCHROEDER: Let's go off the record.

THE VIDEOGRAPHER: Going off the

record 12:22 p.m.
(Recess taken.)
THE VIDEOGRAPHER: Back on the record it's 12:23 p.m.
BY MR. SCHROEDER:
Q. Dr. Nicholson, you said they are not the right graphs; is that right?
A. The graph I am looking at has data missing and relating to the smoking and

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nonsmoking prevalence of X-ray abnormalities greater or equal to two slash one.
Data existed for those groups, I believe, beginning at 25 and certainly at 35 and 50 years.
Q. This is your figure number one; is that right?
A. I am looking at figure number one.
Q. Apart from that, are these otherwise accurate?
A. Yes.
Q. And these two pages of Exhibit Number 3 relate to Lilis' 1986 study, right?
A. Yes.
Q. I want to direct your attention to figure number two.
A. (Witness reviewing.)
Q. What you have done here is a prevalence analysis for smoking versus nonsmoking risks for asbestosis based on the data in Lilis' 1986 study, right?
A. Yes.
Q. The 1986 cohort of workers, those are insulators; is that right?

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MS. DIX: Objection to the form of the question.
A. Yes.
Q. And they were drawn from the same cohort that the 1981 to '83 people were drawn from?
A. No -- oh, yes, they were. There are different groups, though.
Q. Those are the New Jersey and New York insulators in the '86 study; is that right?
A. I believe so. I think it's the 1117.
Q. The results now - you don't report Lilis' '91 study in your report, do you?
A. I don't believe so. I don't know. I have to look at my report.
Q. I will ask, Mr. Kazan, if he would hand you a copy of your report and if you can tell me whether or not you do report on Dr. Lilis' 1991 epidemiological study?
MS. DIX: I think it's 1981.
Before --

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MR. SCHROEDER: 1991.
MS. DIX: Before you said 1981.
A. No, I just have her 1986 study.
Q. Would it be fair to say, Doctor, the analysis you are drawing from the 1986 study of Lilis are representative of the similar kinds of analyses you would draw from the 1991 Lilis group?
MS. DIX: Objection to the form of the question.
A. I believe so. I would have to look at the two papers.
Q. Do you have any reason to believe that the analysis would be substantially different?
A. I don't believe so, otherwise I would have --
MS. DIX: Objection to the form of the question.
A. I don't believe so.
Q. Figure number two reports the prevalence of asbestosis among smokers and nonsmokers in Lilis '86, right?
A. Yes.
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Q. And you have got circles or black dots, those are the point estimates from the study, right?
A. Yes.
Q. Then the lines that go vertically up and down with a bar at the bottom and an arrow on some at the top are confidence intervals, are they not?
A. Yes.
Q. What this means is -- well, you tell me, what's the definition of a confidence interval?
A. It's the range over which one can be at least with 95 percent confidence - 95 percent confidence that the actual value would log.
You know there is uncertainty about this particular measurement, name it six, but in another equivalent study could be four, could be eight, could be two and that's because it's very small numbers.
I think there were two points in one of the two categories in that lowest level.
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Q. So when you are looking at a study and it reports point estimates, you also need to look at the confidence intervals, do you not?
MS. DIX: Objection to the form of the question.

A. In most circumstances there is some information on that worthwhile.
Q. Yes.
What, if anything - figure two shows, does it not - is that for asbestos exposed and in this case insulators, who have less than ten years from onset to asbestos, the smoking risk was almost - the risk ratio was almost six, right?
A. It was six for that particular group, but there is a huge uncertainty on it, because of the small numbers and that is discussed in the paper and secondly, the finding of the small number of - small percentage of abnormalities there still raises an issue. There could have been one or two of those individuals exposed other than as an insulator; that point is highly uncertain.

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Q. It's unusual to find people with fewer than ten years of exposure with a higher risk --
A. It's not a high risk, it's comparing --
Q. -- just under ten years?
A. Well, no, I'm sorry - to have a level approaching 20 percent is unusual, yes.
Q. Then if you go from ten to 20 years since first onset, you report from Lilis a risk of about 2.42 with a confidence interval that goes down just above one, right?
A. Yes.
MS. DIX: Do you want him -- you are referring back to --
MR. SCHROEDER: He seems to remember the data. If he wants his report --
MS. DIX: If you want to see your report --
THE WITNESS: That's fine.
Q. Then from 20 to 30 years since first onset, the risk reported from this study, the risk ratio for smokers versus

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nonsmokers for prevalence of parenchymal abnormalities includes a confidence level of one, right?
A. Yes.
Q. And if you look at 30 to 40 it includes one as well, does it not?
A. I am not sure about that.
Q. If it doesn't include it, it's almost right on top of it?
A. It's very close to it.
MS. DIX: Objection to the form of the question.
Q. Then 40 to 50 includes one as well, does it not?
A. That's correct, but I indicated in

the report the inappropriateness of considering the 40 to 50 group as a measure, as a real measure of smoking, nonsmoking effect, because we are dealing, with this case, a measured effect at the one slash not level that is approximately 80 percent, so obviously you can only have a very tiny difference between nonsmokers and smokers and that is why for the years from onset, from 25

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onwards, one wants to look at the same ratio for X-ray abnormalities of two slash one and higher.

Q. Now, judging just though from figure two which relates solely to one over zero finding, correct; is that right?

A. Yes.

Q. Judging just from figure two, figure two tells us, does it not, that once you are past 20 years from first onset of asbestos exposure, one cannot say based on the 1986 Lilis study that there is a statistically significant difference between smokers and nonsmokers?

A. Yes.

MS. DIX: Objection to the form of the question.

Q. Isn't that correct?

A. No, it's not correct.

Q. Why is that?

A. Because you can combine, for example, just the data from the 25 year and the 30 year and you have a confidence limit that is well above one.

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You can combine 15, 25 or 35 and the confidence level is well above one again. You would have a level of roughly two and-a-half and I don't know what the confidence level would be, but it would be something I would estimate to be 1.5 or greater.

Q. If I have a group of people who are on average 25 years, let's say, since first onset, figure two would tell us that the confidence interval for that group would embrace background, does it not?

A. Not necessarily, it depends upon the size of the group. I indicated that when you are looking at these various groups, the confidence level is higher; as you get more people it increases. There is a great similarity between 15, between 25 and 35.

Q. You did not in your report combine the group of 20 plus years since first onset all the way through 50 to see whether or not you get a statistically significant result, did you?

MS. DIX: Objection to the form of

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the question.
A. Let me see my report. I think
there is --
Q. You need to look at Mr. Kazan's
copy.
A. (Witness Reviewing.)
Q. I would direct your attention, I
think, to page 17.
A. I have it. They are combined on
page 13 and it includes the point of 50 years,
the ratio was 1.6 and the confidence limit on
that would be .3 or .4 and --
Q. That's what I'm asking you, but
there is no confidence interval here
calculated for that 1.6?
A. They are not calculated for that.
I am telling you what it roughly would be
looking at the data on this graph.
Q. Are you telling me without running
the calculations that you are confident that
the confidence interval would be above one?
A. Yes, indeed I am saying that very
strongly.
MS. DIX: Objection.
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Q. Have you run that calculation?
A. No, but I know that the answer I
gave is correct. When you have a five
confidence limit as they are, even though two
of them are on the line and one a tad below,
putting them altogether is going to have a
confidence limit that is well above one.
Q. How far above one?
A. I can't say that. I think it
might be -- it's probably -- even with this
50 point it would be .2, .3.
Q. So you are saying about 1.2, 1.3;
is that right?
A. Yes.
Again, you are looking at the
bottom of a point that is one that should not
be considered even in the ratios and I
described the problems therein and the
solution to them which was produced in the
report showing that at the higher level there
indeed is again a twofold increase risk of
abnormalities between smokers and nonsmokers
and the X-ray category two slash one and
higher. If you don't have it on the graph you
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have it in the text.
MS. DIX: Do you have an extra
copy to mark for the record?
MR. SCHROEDER: I don't have one.
MS. DIX: Do you want to take a
break now for lunch?

MR. SCHROEDER: It's up to you, Doctor.
THE WITNESS: Is there more on this particular issue?
MR. SCHROEDER: If you want to go through this issue then take a break.
THE WITNESS: Let's finish this issue.
Q. Would you agree, Dr. Nicholson, this 1986 study of Dr. Lilis concludes that once you are 40 years from onset of exposure for those who make it that far, that there is no statistically significant smoking effect?
MS. DIX: Objection to the form of the question.
A. I would not agree with that.
Q. If I can direct your attention to your report, page 17 and ask you a question.

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A. (Witness Reviewing.)
Q. You provide a chart from Dr. Lilis' paper where it says 40 plus years from first exposure; do you see that?
A. Yes.
Q. Over on the right on the P value it says NS, which has a footnote says not significant?
A. That's correct.
Q. Why?
A. But this report reports not only on the abnormalities that are one slash not or greater, it also reports on the abnormalities at the two slash one. They show a dramatic doubling in that point and if you combine the data of this one, even, you know, it's low forced by the broad exposure circumstances, the combination of a -- of the 45 to 50 year point or whatever it is at one slash not and that at two slash one would have that reach of 40 plus with a statistically significant difference from one.
Data for that point are not present on the graph, in these graphs -- it's

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right underneath the table. At such a high level -- I will read it at 40 years. Both smokers and nonsmokers have more than 75 percent of their X-rays abnormal. At such a high level differences are required to be small in the overall comparison that is in this ratio.
However, in that time from onset category a substantial smoking related effect exists in the percentage of individuals with profusions of 1-2 slash one or more.
15.4 percent for smokers and 7.7 percent for nonsmokers, so there is a twofold effect and that - plus this would have the combined effects certainly having a

significant -- a confidence level above one given that I'm going by the confidence level for the one slash not, which just touches the line and makes that point --

Q. Let me --

A. -- Nonsignificant, although I think my estimates of the confidence limit are more stringent than Lilis'. She has all points except the last one, the significant

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and I have the bar extending below the line for one of them.

MS. DIX: For ease of reference, at the break I think maybe we should get a copy of the report and mark it as Nicholson 4, is that okay?

MR. SCHROEDER: That will be fine.

Q. Dr. Nicholson, if you were to take the two slash one group out of your answer --

A. That group was very important.

Q. I understand that, but if you take them out for a minute, would you then agree - is that at 40 years since first onset Lilis reported no smoking effect?

MS. DIX: Objection to the form of the question.

A. That's correct, because the level of abnormality for abnormal people is virtually close -- well, it's 75 percent. You are at such a high level that any ratio has to be small and the small numbers required by spreading the data out across five exposure categories, you have an uncertainty that does not exclude one.

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Q. So put another way, is it the same thing as saying that if you are going to measure on the one over zero cutoff, that once you get to 40 years since first onset, whether or not you smoke doesn't have a statistically significant response?

MS. DIX: Objection to the form of the question.

A. In those limited data that happens to be the case, but these data nevertheless at that point indicate a clear smoking effect.

Q. Well, --

A. And you have to look at all the data. You don't hide your head in the sand and say I am going to take this little snippet, because for a totally absurd unrelated reason it's required to, in essence, show no effect.

Q. You are saying the two over one needs to be considered?

A. Absolutely.

Q. Have you looked at the trust scheme for compensation?

A. I may have at some time, but it's

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been some time ago.
MS. DIX: Are you referring to the TDP?
MR. SCHROEDER: Yes.
A. I've seen it and I vaguely recall it, but don't hold me to the details.
Q. Do you know what the ILO cutoff rating is for the trust to compensate for what's known as a category two asbestosis claim?
MS. DIX: Objection to the form of the question. You are not characterizing the terms correctly, Tom.
Q. That's fine.
Do you know what the ILO rating is.
MR. KAZAN: It's not fine, the trust uses the term bilateral interstitial disease.
Q. Do you understand what the ILO rating is that the trust uses for a threshold for bilateral interstitial disease under the TDP?
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MS. DIX: Objection to the form of the question.
A. I don't recall exactly, but we are not making - that particular issue doesn't play a role in looking at whether there is a cigarette smoking effect or X-ray abnormalities.
Q. What particular issue?
A. Whatever the trust compensates for.
Q. You are saying -- what do you mean by that?
A. I'm saying whatever the trust compensates for plays no role whatsoever in data showing a smoking effect on X-ray abnormalities.
Q. So you are saying they are not necessarily related to each other?
MS. DIX: Objection to the form of the question.
A. They are dealing with two different things. One is answering the question of who should be paid and the other is what is the effect of cigarette smoking.
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Q. Fine.
Why don't we see if we can finish out this section and take a break, is that all right?
A. Yes.
Q. If you take a look at the other

figure that you have there, Dr. Nicholson, which is figure -- yes, figure number one?
A. (Witness Reviewing.)
Q. Doesn't figure one show us that for people exposed in this Lilis 1986 cohort, that if there is an increase prevalence of smoking among asbestonics, that what you are finding is it's being reported earlier than when it otherwise would be reported, based on years from onset, if you didn't smoke?
MS. DIX: Objection to the form of the question. Do you understand that?
A. It's -- what it shows is that it's evident at every degree of asbestos exposure, so the smoking effect is manifest early and it's manifest at lower asbestos exposures.
Q. But doesn't this figure one also tell us that given sufficient time, if you use

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the one over zero as a cutoff, that even if you don't smoke, you are going to have roughly the same level of abnormality?
MS. DIX: Objection to the form of the question.
A. No, I didn't say that at all. It says that as you progress in time, the minimal abnormalities will be found in smokers and nonsmokers and when you are close to the hundred percent in both groups.
This does not show that there is a much higher degree of abnormality in the smoking group compared to the nonsmoking group at 50 years. I am speaking about - there are far more people with severe asbestosis present in the smoking versus the nonsmokers, so when you in essence come to a dead-end because of saturation, the measurement criteria, the one slash not criteria, you then have to see if there is evidence of parenchymal disease of a greater nature and that's what I did.
Q. And that's what I want to separate those two concepts, if we can just for a minute and I understand you think the two over

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one group is important, but I would like to separate them and talk about them for just a minute.
If you look at just the one over zero and the question is who in this group has more evidence of parenchymal abnormality as judged by something greater to or equal to one over zero, isn't it true that over time eventually, if you can be fortunate enough to be 50 years -- live to be 50 years from first onset as an asbestos worker, that both the smoking asbestos workers and the nonsmoking workers are essentially the same when judged by one over zero?
MS. DIX: Objection to the form of

the question. You are presenting a hypothetical that's taking out a clear factor that the doctor testified needs to be there.

MR. SCHROEDER: That's fine.

Q. Am I not correct?

A. You need data saying asbestos is sufficiently bad. Both smokers and nonsmokers face a disaster, if workers, insulators, live

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40 or more years from onset of exposure.

Q. If the disaster is measured?

A. But there is a greater disaster for those that smoke than those that do not.

Q. I understand that.

A. They are very clear.

Q. Then if you want to define the disaster as being simply anything greater than or equal to one over zero without defining how bad a disaster it is beyond that --

A. I don't. Do you want to be so limited?

Q. I want to ask you the question, that's my prerogative. I can ask you that.

If you want to define the disaster as anything at or greater than one over zero, it's equally as bad for everybody once you are 50 plus years?

MS. DIX: Objection.

A. No, it's not. The chances are very high that it is greater than for the smokers rather than for the nonsmokers. They are not sufficiently high that one can say at the 95 percent level there is a 90 percent

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chance that it is higher for the smokers compared to the nonsmokers, so I'm moving that confidence level up.

Q. So at 95 percent they would be the same, roughly?

A. No, they are not the same, they are clearly different, but there is a - there is a five percent chance they are the same, but in '95 percent that the smokers are higher, roughly.

Q. I'm sorry, Doctor, I thought you just said at the 95 percent confidence interval you couldn't say there is a difference, but at the 90 percent interval there would be a difference?

A. At 95 percent what you do have is a five percent chance that they might be equal, but that means since the 95 percent is right on the line, for the statement I am making it on the line, that would put it that the chances are that smokers - there is a 95 percent chance that smokers indeed have a greater abnormality than nonsmokers, that's a pretty strong statement about a smoking

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effect, without adding the very definitive information I have already mentioned.
Q. And the two -- we'll leave that aside.
MR. SCHROEDER: Let's take a lunch break.
THE VIDEOGRAPHER: Going off the record it's 12:53 p.m.
(Luncheon recess taken at 12:53.)

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A F T E R N O O N S E S S I O N
(Time noted: 1:38 p.m.)
W I L L I A M J. N I C H O L S O N, Ph.D.,
resumed and testified as follows:
MR. SCHROEDER: Let's mark this as Exhibits 4 and 5.
(Nicholson Exhibit 4, Dr. Nicholson's report, marked for identification.)
(Nicholson Exhibit 5, Graph, marked for identification.)
THE VIDEOGRAPHER: We are back on the record. It's 1:38 p.m.
BY MR. SCHROEDER:
Q. Dr. Nicholson, before lunch you had made reference to your report and we said we would mark it as Exhibit 4. Just for the record can you now confirm that what has been marked as Exhibit 4 is your report in this case?
A. Yes, it is.
MS. DIX: That's in the Falise 1 case?
MR. SCHROEDER: It was in Falise 1

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and 2, actually.
Q. Dr. Nicholson, I would like to explore with you some questions about the prevalence of studies on asbestosis, okay, sir?
A. (Witness Nodding.)

Q. Would you agree that the prevalence of studies that are sighted in your report for parenchymal abnormalities rarely give information on asbestos exposures in the separate groups of smoking versus nonsmoking?
A. That's correct.
Q. Would you also agree that if you have a prevalence study that uses a one slash zero as a lower threshold for parenchymal abnormality, that what the prevalence study tells you is the increase in prevalence for those at the one slash zero goes all the way to the three over three?
MS. DIX: Objection to the form of the question.
A. Yes.
Q. And that the prevalence studies do not then tell you whether because of smoking

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you would move simply from a zero slash one to a one over zero, would you agree with me?
MS. DIX: Objection.
A. I don't understand your question.
Q. The prevalence studies measure prevalence within a range or spectrum as defined by whatever ILO category is used in the studies, correct?
A. That's correct, it can be from one slash zero and up. It's likely to be limited to the lower range in most circumstances.
Q. But the prevalence studies do not speak to whether smoking moves you from a zero over one category to a one over zero, that limited issue studies don't speak to that, do they?
MS. DIX: Objection to the form of the question.
A. They just indicate that they are a greater percentage of one slash zero in one group versus the other.
To the extent that there are - that smoking has increased that, there is a likelihood that some of those that were one

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slash not and smoking might have been zero slash one, that's the logical precursortude to one slash not, but what the underlying reading would be in the absence of smoking, we don't have that, because -- other than in another group.
Q. Right and because we don't have that underlying reading in the absence of smoking, the limitation of a prevalence study is it can't tell you whether or not because of smoking you would move from that zero slash one simply to a one over zero?
MS. DIX: Objection to the form of the question.
A. On an individual that can be

determined, but the likelihood is that in the population that in fact is what is taking place.

You have people that would have had zero slash one had it not been from the smoking. What is the alternative to an increase is an increase from a lower level.

Q. But what I want to focus on is not just an increase from one, let's go from an

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increase from zero over one to anything higher than that.

I want to know whether the prevalence studies will average the limited question of whether you will go simply from a zero over one as a nonsmoker exposed to asbestos to a one over zero, they don't directly answer that?

A. They don't directly, but more likely than not that is the case.

Q. But the prevalence studies don't give us those values, do they?

MS. DIX: Objection, asked and answered.

A. They don't -- that's not a measurable thing in a prevalence study. You are looking at what's there. What's there has to come -- what's there is different in one group versus another.

What is the logical state absent the smoking and that would be a state similar to the nonsmokers, so there would be more zero slash one and less one slash zero, so you could analyze that particular feature in the

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two groups, but common sense indicates to you that it is likely, certainly more likely than not, that some of the individuals in the smoking group would have had zero slash one absent their smoking habit or maybe zero slash zero even.

Q. But the prevalence studies won't tell us whether you go from a zero one simply to a one over zero, you are saying, if I understand your testimony, that it will tell you whether or not you go from a zero one to somewhere in between one over zero to three over three in the range of prevalence, right?

MS. DIX: Objection.

A. In most of the studies, the range is probably - from abnormalities would be one slash zero to maybe a one slash two. I doubt there would be maybe one or two here and there are abnormal X-rays at the two level.

The exposure of the groups involved with the exception of the insulators have limited asbestos exposure.

Q. If I wanted to know, Doctor, whether smoking makes you go from a zero over

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one simply to a one over zero, but not more, the prevalence studies won't answer this question, will they --
MS. DIX: Objection, asked and answered.
Q. -- by the definition of the nature of your study?
A. Well, they would -- the possibility that one went from a -- well, you can look for a study in which there were no - there were one slash not X-rays, I don't know if that is the case.
If you had such a study than it would be likely that it simply moved you from a zero slash one to a one slash zero.
Q. You would have to have a study that simply had the one over zeros?
A. That's right and they would -- even higher, but the possibility certainly is it does move it higher in particular circumstances.
Q. But what I want -- if I want to focus on what the data shows, if I -- by design these studies measure not only one over
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zero, but anything above one over zero?
MS. DIX: Objection.
A. Correct.
Q. By measuring anything greater than one over zero they don't precisely answer the question of whether it moves you over from a zero one, just to a one zero, right, if you smoke?
MS. DIX: Objection.
A. Well, because of the preponderance of one slash zero it would be indicative that would have been the case in a fair percentage of the cases, so that certainly was happening. Whether it had moved higher or not, I can't say and I don't think anybody can say.
Q. When you say whether it moves higher or not, you mean from zero one to one zero?
A. Yes.
Q. If I asked you the same series of questions about moving from a one zero to a one one, would you give the same answers?
MS. DIX: Same objections to the
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prior series of questions.
A. I don't know there is any evidence of that in the data, that set that I have there.
Q. Would you agree the prevalence analysis do not control for age?

MS. DIX: What studies?
MR. SCHROEDER: Of the asbestosis, the prevalence analysis of asbestosis -- I'm sorry.
Q. The prevalence analysis of the parenchymal abnormalities.
A. Most of them did not.
Q. Would you agree that they also do not control for latency?
A. That's correct, they took the work force as it is, some of them may have eliminated people with less than ten years and the criteria of difference was smoking or not and so there would have to be other factors to be important and to be important across studies you would have to be some selection for those factors, because job -- in circumstances, the job and work experience of

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smokers and nonsmokers to me is very similar.
Q. You assumed in your expert report that the exposures for smokers and nonsmokers were the same, did you not?
A. That's right, because there is no evidence whatsoever to indicate that they would be different, so it's the same work force in character and the only difference being cigarette smokers were separated from nonsmokers.
Q. But you also acknowledge, do you not, that exposure levels to asbestos were higher for older workers than for the more younger workers?
MS. DIX: In parenchymal abnormalities?
Q. Yes, we are still talking about parenchymal abnormalities; do you agree with that?
A. In general that would be the case, but there is no reason -- in heavily exposed workers, there can be a slight change and that leads to the nonsmokers having a longer period of work on average, so that would make the

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nonsmoking group of an older age than the smokers group, because the smokers are killed off by their smoking and thus correcting for any age effect would increase the magnitude of the ratio.
Q. Well, let me see if I follow that.
Older asbestos workers generally were exposed to more asbestos, true?
A. Yes.
Q. Of the asbestos workers who are in the survivor cohort, those who were around when you made your studies, if they smoked, and were older workers, they tended to be more heavily exposed to asbestos, right?

MS. DIX: Objection to the form of the question. Which studies, which cohort?
MR. SCHROEDER: It's a general proposition.
A. Older workers have more exposure than younger workers.
Q. We established that, did we not?
A. Yes and that's what you are saying

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again.
Q. I'm saying the smokers tend to be the ones who are older workers?
A. No, the nonsmokers are - preferentially the older workers compared to the smokers are more nonsmokers than smokers in a population, because the death rate of smokers is higher than nonsmokers, so they continue to work, they don't get carted off to the morgue from their smoking habit and thus it has been established that the nonsmokers tended to be older and tended to have worked longer and thus had a greater exposure.
Q. So if you want to measure smoking effect, you need to put the smokers who died back into the cohort?
A. Not necessarily.
Q. Well, if the question is - let me ask it this way: If I were to ask --
A. That would even make it worse, again, because then you have got many more cases of lung cancer.
Q. The question is after 40 years as a smoker are you anymore likely to have a

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parenchymal abnormality based on a one over zero threshold?
MS. DIX: Objection to the form of the question.
Q. The answer will, if you consider smokers or nonsmokers, the answer will both be they are equally to have a one over zero?
A. Not necessarily.
MS. DIX: Objection to the form of the question.
A. It depends on the exposure circumstances. If you are talking about a group of insulators, yes. If you are talking about any one -- virtually any one of the other studies, no, because the level of exposure is insufficient to have most of them with abnormal X-rays; I mean there can be others. I know most of them were exposed to asbestos and insulators --
Q. If you took the prevalence studies in your report on your table seven, take a look at Exhibit 4, it's on page 12.
A. (Witness Reviewing.)
Q. You see those there, right, sir?

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A. Yes.
Q. Your table seven, those are the studies upon which you draw your conclusion of a 50 percent prevalence of smokers over nonsmokers for parenchymal abnormalities?
MS. DIX: Objection to the form of the question.
A. That's part -- that's a substantial part of the analysis, but there are other studies as well that are discussed following it that come to the same conclusion.
Q. If you take the studies listed in table seven and the others in your report that speak to the issue of the smoking, nonsmoking prevalence for parenchymal abnormalities and you were to go in those studies and stratify them for those people exposed to greater than 20 years of asbestos, would you agree with me that you would find there is no statistically significant smoking effect?
MS. DIX: Objection to the form of the question.
A. Absolutely not.
Q. Have you done that analysis, sir,
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stratifying?
A. I haven't done it for every study. In some cases you see here is language lines with the 40 to 60 year age group and there is an increase to prevalence in smokers compared to nonsmokers and where it was available -- Lilis, it's there.
Each group contains people, virtually all groups contain people, many people with more than 20 years of exposure and in your hypothesis after 20 years the whole smoking thing goes away, that means the smoking effect must be enormous in the earlier years.
Q. Or it could mean that it's very poorly measured, couldn't it?
MS. DIX: Objection to the comments.
A. No, not at all. When you consider over 20 studies and see this definitively expressed in virtually all of them, you are not going to have that many investigators quote/unquote performing bad analysis or whatever you just said.
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Q. Have you stratified these studies based on those that report information so that you can determine an effect for people exposed for greater than 20 years and compare the smoking?
MS. DIX: Objection to the form of

the question.
A. It is not possible to do so. When it was I did that and that was the analysis of Lilis. It was very clear -- it was very clear that it was continuing through all exposure categories and you see it not only causing an increase from a zero one to a one zero, making it abnormal, you see it changing what would be a lesser degree of abnormality to something that's very substantial, two one or greater. The effect is taking place very clearly at times beyond 40 years from first exposure.
MO MR. SCHROEDER: I move for the record to strike after the response of yes or no.
Q. If you stratify these studies based simply on a one over one ILO - let me ask it this way: Most of these stratify

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based on greater - to whether it is greater than or equal to one slash zero, correct?
A. Yes, that's what the information had available.
Q. And what I am asking is if you were to stratify on a one slash one ILO rating - have you done that?
A. When it was available, yes.
Q. And?
A. But it's available only in three or four, I don't remember the number. It tells you what the number is here. There were two studies using one slash one and one -- there were two studies that used one slash one as the lowest level of abnormality and two studies also required competent presence of clinical sciences of pulmonary function deficits, so it looks like four were dealing with the one slash one level.
Q. Do some of the studies that report on a lower threshold of one slash zero also contain data that you could then stratify yourself at a one over one?
A. No.

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Q. Have you attempted to do that?
A. It wasn't there.
Q. Okay, is it fair to say you tried and did not find it?
A. I looked for it I believe in all studies, but to my awareness there was -- the abnormalities were based on one slash not. Virtually every study had only one table according to abnormalities.
Q. Let's talk about the disease of lung cancer, Doctor.
Is there a threshold exposure for asbestos of lung cancer?
A. I don't know of one.
Q. For a group of individuals who

would have had 20 years of an asbestos exposure or more, would it be your opinion that asbestos would be a substantially contributing factor to lung cancer?

MS. DIX: Objection to the form of the question.

A. It depends on the intensity of the exposure and how you define substantial. Certainly there is a big difference between

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20 years as a garage worker and 20 years as an insulator.

Q. Are you saying there is a threshold under which smoking - under which asbestos would not be a risk for lung cancer?

A. Not at all. You are asking about a substantial risk and that's a fuzzy term and I'm making the point that you can have a modest increase in risk for garage workers and in my terminology substantial risk for insulators.

I don't know if a five percent risk may be characteristic of a group of garage workers would be called substantial or not. Certainly it would be for those who develop the disease, but it's certainly present, so it's a linguistic thing.

Q. After 20 years of asbestos exposure, what would be the relative risk for lung cancer for a garage worker; is that who you were talking about?

A. Yes.

A typical garage worker in New York City who does one to two jobs a week,

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brake repair, you are looking at ballpark - I mean one percent increase in lung cancer, I mean with levels.

Q. One percent lifetime?

A. Yeah over what it would be otherwise.

Q. Would it depend on what garage they worked in as to what the risk would be?

A. Yeah, it would depend on the number of jobs he does. Basically -- well, the average exposure for doing a job is about half a fiber per milliliter, so you are exposed for two hours say and for a car job you would then have something like a ten to two tenths -- say two tenths of a fiber for that day. If you do two jobs a week, so that's two tenths of a fiber -- what did I say -- started with two tenths of a fiber for a day, that gives you four tenths of a fiber day for a week and that brings you back down to one tenth for a week and then you multiply that by 50, so you are at -- I'm sorry. I don't want to go that way.

Over the week, two cars with brake

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jobs, we came up with a tenth of a fiber continuous exposure for a week, so extend that for a year, in ten years you will have increased the risk of lung cancer by one percent.
Q. For a ten year period, is that right?
A. No, at the end of that ten-year period, his risk of developing lung cancer would be one percent higher than what it would have been if he had not worked at the garage doing a couple of car brakes a week.
MS. DIX: Is this a smoker or nonsmoker?
MR. SCHROEDER: I object to the commentary; his answer is what it is.
MS. DIX: I'm clarify. It's been a while since the question.
A. That's how much it's multiplied, whatever the underlying risk is of that - of a smoker or nonsmoker. Mortality, -- small risk you can see an increase in abnormal X-rays; one braker who has been employed at a garage for 20 years, but you only see it in
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the ones that have done a lot of jobs.
Q. If you had three garage workers exposed for 20 years and all three had lung cancer and if they were exposed under the circumstances you just described, would it be your opinion that asbestos was a contributing factor to their lung cancer?
A. It contributed to their risk of lung cancer, other factors contributed more.
Q. Contributed more?
A. Yes.
Q. How do you know there were any other factors?
A. Well, there is certainly something contributing to our -- the individual's risk of lung cancer, be it biological factors within the body, be it material in the air we breathe.
There is a background risk of lung cancer in the general population and that's increased in this particular circumstance talked about by one percent, so something else is more likely the cause of lung cancer than doing two brake jobs a week in the auto
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industry.
Q. Would you agree that once a person gets asbestosis, that your risk of developing lung cancer is increased?
MS. DIX: Objection to the form of the question.

A. Obviously it's increased over someone that has not had any exposure to asbestos and the presence of asbestosis indicates substantial exposure, so there is -- it's corresponding. Lung cancer increases -- if you are asking separately does asbestos itself separate from exposure increase lung cancer, is that your question?
Q. No, my question is there seems to be data that says that once you have asbestosis, your risk of getting lung cancer is increased above anyone else otherwise exposed to asbestos who has not yet developed asbestosis?
MS. DIX: Objection to the form of the question.
Q. Is that correct?
A. It depends. If you are

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considering all other people without asbestosis, then those with asbestosis would have a greater risk of lung cancer, because they have a greater exposure.
Q. What approximately is that increase risk in lung cancer once you have asbestosis?
A. You begin to see asbestosis at 25 fiber years per cc. At that level you have got a 25 percent increase risk of lung cancer and others -- in fact you have got measurable asbestosis at higher exposures, so you are dealing with a fair amount of exposure and thus a fair lung cancer risk.
Q. Some studies report that the risk of lung cancer, once you have asbestosis, can be in the range of eight and nine, do you agree with that?
MS. DIX: Objection to the form of the question, vague.
A. The asbestos related risk is eight to nine times with asbestosis that which it would be otherwise?
Q. Yes, sir.

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A. You have to have someone working in a textile mill at 45 to 50 fibers per milliliter year, at that risk. You are looking at somebody with at least twice the exposure of insulators, such people are rarely seen.
Q. What is your definition of the word synergy as it applies to the relationship, if at all, between smoking, asbestos and lung cancer?
A. I would apply synergy to an interaction that is different from a simple additive interaction.
If the effect of -- when the effects of each agent is different, from the

sum of the effects when the are termed a synergistic interaction, most circumstances we are speaking of here we are looking at a multiplicative interaction between asbestos and cigarette smoking, although you have interactions that are less than fully multiplicative.

You needn't have imagine an interaction where two exposures give rise to a

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lesser risk and that one agent creates a protective effect. I think synergy can apply there as well, so we are just defining the term.

Q. In terms of the studies that address lung cancer and smoking in asbestos interaction, those are statistical studies, right?

MS. DIX: Objection to the form of the question.

A. The majority of them are, yes.

Q. And the inferences to be drawn from those studies are inferences of whether they are statistical interaction, correct?

MS. DIX: Objection to the form of the question. You understand what he is talking about statistical interaction?

A. I don't understand that.

Q. You would agree, would you not, Dr. Nicholson, that you used the phrase interaction before, did you not?

A. Yes.

Q. And by interaction, you meant a statistical interaction?

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MS. DIX: Objection to the form of the question. Objection, misstating prior testimony.

A. I used the term interaction, but actually the interaction between two agents is obviously biological. You can have evidence of the manifestation of events and the description of that manifestation would be statistical in nature.

Q. It would be drawn from the statistical studies?

A. Yes.

Q. And the phrase synergy means -- I think you said biologic interaction, right?

A. I said it means an interaction that leads to effects from two agents or more, let's leave it at two agents that are different from what would be the sum of each one acting alone.

Q. What I want to do is see if you agree there is a distinction between statistical interaction which would be whether there is any departure from whatever model you define, additive, multiplicative on the one

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hand, versus the concept of something occurring at the biological level?
MS. DIX: Do you have a specific question that you want to pose to the doctor?
MR. SCHROEDER: That was a question.
Q. Do you agree those are two distinct concepts; do you understand what I am asking?
A. Not really, but what one observes and does so and establishes with statistical analysis, an effect that suggests some biological interaction of unknown character no way indicates what it is.
Q. While it may suggest some biologic interaction, whether or not biologic interaction occurs is a phenomena that's been termed as synergy, right?
MS. DIX: Objection to the form of the question.
A. I guess most circumstances can be -- yeah I will agree that if there is an interaction that takes place, that plays a
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role in the manifestation of disease, that interaction can be called synergistic.
Q. By calling it synergistic, that means something happening at the biological level?
A. Yes.
Q. So if something is happening at the biological level in fact then we call that synergy, is that your testimony?
MS. DIX: Objection to the form of the question.
Q. If there is interaction at the biological level you call that synergy?
A. If it's manifested in a difference, an observable phenomena, there is something there that is totally unrelated to any anything we were measuring, you can't call it much of anything, because we don't even know if it's taking place and there can be interaction at the biological level that are unobservable and I don't know what I would call them.
Q. If there were interaction at the biological level that science doesn't yet know
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yet what to call, as you said they would be unobservable?
MS. DIX: Objection.
A. If they have been -- I mean you may have not used the right tools to observe them. I'm indicating that you can have things

happening that we don't know about.
Q. And it's true and I think you say in your report that to date there is no scientific consensus on any biologic mechanism for interaction, correct?
A. There is no consensus, but there is a lot of work going on in that area and certain risks of cigarette smoking and asbestos. There is considerable data out there and some hypothesis that people made, some may disagree with them, some are -- there is general agreement, but we don't have an ideal model at this point.
Q. I believe you said in your report that if there is a mechanism by which asbestos and smoking interact, it's not fully understood presently?
A. That's correct.

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Q. Would you agree, Dr. Nicholson, that most epidemiologic statistics outside the area of asbestos and smoking were based on assumed multiplicative relationships?
MS. DIX: Objection to the form of the question.
A. I can't answer that. I haven't made a survey that would determine whether most are or most are not.
Q. Do you consider yourself an expert in the area of epidemiology?
A. In certain aspects of it, yes.
Q. Do you find -- do you know who Rothman is?
A. Yes.
Q. Do you consider Rothman to be authoritative in the area of epidemiology?
A. He carries some authority. There is some things he does I disagree with.
Q. As applied to issues of interaction do you find them to be authoritative?
MS. DIX: Objection, vague.
A. I can't make a statement one way

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or the other on that. I would have to look at his writings on it. I've read them, but I just don't recall the details now.
MR. SCHROEDER: Let's mark this as 6.
(Nicholson Exhibit 6, Synergy between asbestos and smoking on lung cancer risks, marked for identification.)
Q. I'm going to take an exhibit out of order.
Let me hand you what's been marked as Exhibit 6. It's an exhibit that was produced to us when we got the response to the subpoena.

A. Yes.
Q. From you.
Do you recognize that as a copy of the Ehren article from your files?
A. Yes.
Q. That was sent to you by plaintiffs' counsel in this case?
MS. DIX: Objection.
A. I don't know.

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Q. It appears to have been faxed to you by Orrick, Herrington.
A. Yes, it would appear to be so.
Q. If you would, do you recognize that to be a review of the studies on the relationship between asbestos and smoking for lung cancer?
A. Yes.
Q. Do you find that review to be authoritative?
A. No, I disagree with it.
Q. Why?
A. I believe there are four studies that discuss the issue in review here. The measure of synergy that he uses is one that depends on the magnitude of exposure when you get different measures of synergy, depending upon the reason of exposure, not in terms of degrees of interaction and I comment on that here someplace.
As he describes synergy he uses the term attributable portion and for a relative risk of five, the attributable portion is 72 percent in this case.

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For a directly multiplicative interaction, the attributable portion falls to 45 percent of asbestos; relative risk is two and down to 30 percent; the asbestos risk is 1.5, so you get -- it presents a poor measure of interaction and it doesn't take into account the -- this feature.
Q. By using an attributable proportion, Ehren ends up assigning all of the synergistic risk to smoking, does he not?
A. I mean the synergist involves two agents.
Q. Correct.
MS. DIX: Do you need the article back?
Q. If you use the attributable proportion analysis and limit yourself to that, by definition, don't you assign all of the excess risk from the joint effect to one factor such as smoking?
MS. DIX: Objection to form.
A. I would hope not.
Q. That would be - would that be inappropriate?

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A. Yes.
Q. Why is that?
A. Because an interactive effect involves two agents.
Q. Is that why you used the Chase Kotin model in your expert report?
MS. DIX: Objection to the form of the question.
A. Yes, because that does that -- you can separately calculate the proportion of the risk attributable to asbestos and that to smoking and they are semantic in terms of formulation and if you had equal risk -- you would have -- equal singular risks for smoking and asbestos, you would end up with equal proportions of attributable risk.
Q. In your report, you advocated adopting an allocation of the excess risk on a proportional basis to asbestos and smoking, right?
MS. DIX: Objection to the form of the question. Are you talking about lung cancer?
MR. SCHROEDER: Yes.
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A. If you wanted -- if you calculate that attributable risk, yes.
Q. You think that's a fairer way to do it than what Ehren did?
MS. DIX: Objection to mischaracterizing prior testimony.
Q. I'm asking you?
A. I believe so.
Q. Would you agree, Dr. Nicholson, that even doing it the way Chase Kotin did is an arbitrary allocation?
MS. DIX: Objection.
A. It's not -- it is a direct result of utilizing a directly multiplicative model.
Q. Sure, but the allocation of the excess risk on a proportional basis is an arbitrary allocation, is it not?
MS. DIX: Objection to the form of the question.
A. I think it is the most logical allocation. You consider each agent acting similarly and to ascribe similar allocation to them relate in accordance with their individual risks.
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I think what is totally arbitrary is to suddenly assign all to smoking or all to asbestos in a joint circumstance.
Q. Are there studies, Dr. Nicholson, that address whether there is interaction between asbestos and smoking for lung cancer

at low levels of asbestos exposure?
MS. DIX: Objection, vague in terms of what low levels means.
Do you understand the question?
THE WITNESS: Yes, I understand the question.
A. I'm looking for interaction studies. In principal the exposure level of asbestos would be low. Yeah, shipyard employment, Blott studies would be one and I'm sure in the array of available studies there are others that would have modest cigarette exposures.
S. Raff studied a cement plant, that's fairly modest. I mean its exposure is certainly much lower than insulators.
Q. Who did?
A. Raff, that's how I know what the

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exposure circumstances were. Some of these I don't recall.
Certainly Blott is considering shipyard workers is an average exposure that is relatively low, because those shipyard workers are exposed by virtue of fibers released by insulators and they themselves largely work with asbestos.
Q. So we are on the same wave length and to satisfy Ms. Dix' concern, how do you define low level of exposure?
A. In these circumstances, one to three fiber per milliliter level; one to five say.
MR. KAZAN: Is that cumulative?
THE WITNESS: No, that's intensity average.
Q. How about on a cumulative basis?
A. For a study to be done, you would need cumulative exposures, probably about 25 fibers per milliliter, that gives you a 25 percent increase and you can see twofold differences.
Q. Just so that I understand then,

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you would define a low level of exposure in a lifetime basis to be how much, cumulative?
A. I don't think that's a low level, but I think in order to accurately be able to look at synergistic effects in asbestos and smoking, that's what you are asking about?
Q. Right. What I am trying to determine, Doctor, is you said the Blott study was an example you thought of, an interaction study at a lower level of asbestos exposure, is that a fair statement?
A. Yes.
Q. My question is over a cumulative exposure, lifetime exposure to asbestos, how do you define low level for purposes of

applying a Blott-type analysis?
A. Well, I guess the Blott study, you are looking at cumulative exposure someplace in ten to 25 fiber years. I would have to look at his paper, but considering - I mean that can be a shipyard exposure.
Q. Do you think that analysis can be applied to non-shipyard cohorts?
MS. DIX: Objection to form.

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A. If you have adequate data.
Q. What kind of data would you need?
A. Just in a population you can look at the separate effects of a smoking group and a nonsmoking group and distinguish them so it's a population size that in fact is calculable.
Q. Would you agree, Dr. Nicholson, that in looking at whether there is interaction in an - in any particular study involving asbestos, smoking and lung cancer, that not only did you need to look at the reported estimate of interaction or synergy, to use your term, but you also need to look at the confidence ranges to make sure it's statistically significant?
MS. DIX: Objection to the form of the question.
A. That would be ideal, but in many circumstances, that information has not been presented, so you look at what you do -- what you have and you get a measure of confidence in the results by the concordance of the data across many studies.

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Q. I understand for purposes of public health that you may look at an epidemiological study and even if the results are not statistically significant, you may want to draw certain conclusions in order to improve public health, would you agree with that?
MS. DIX: Objection to the form of the question.
A. That's correct. If you found suddenly a couple of cases of some extremely rare disease that you might associate with somebody swimming in lake X or walking through swamp Y, you would certainly want to start taking action to look at well, first you can readily prevent - tell people not to swim in lake X or walk through swamp Y until we find out what it is.
Often the case is very simple, even if you have limited data, you stop exposure until you get very definitive data.
Q. If you were going to step into a courtroom and testify to a reasonable degree of scientific certainty --

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A. That's more likely than not.
Q. Yes, you would want to know what's more likely than not, correct?
A. Yes.
Q. You would testify to what is more likely than not?
MS. DIX: Objection.
Q. Would you?
A. Yes.
Q. In science, in order to make a judgment that's translatable into the courtroom, would you rely on a study that does not have statistically significant results?
MS. DIX: Objection. Calls for legal conclusion.
MR. SCHROEDER: I'm asking would he rely on it.
A. Am I taking into account the degree to which reliance should be placed?
I earlier today very strongly indicated multiple reasons that there was a clear smoking related increase in X-ray abnormalities for the 40 plus group of Lilis', both from the one one plus and the two plus
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and I attribute that excess in the one one to be a positive one, so it's the roughly 95 percent confidence level that's a pretty good confidence level.
Q. If I have a study of asbestos smoking interaction for lung cancer and apply 95 percent confidence level and if the additive model falls within that 95 percent confidence level, would you agree that from a scientific point of view you cannot exclude the additive model under those data?
MS. DIX: Objection to the form of the question.
Q. Did you follow that?
A. If this particular case had the 95 percent confidence level right on the line of what would have been an additive model, you can state very strongly that is the case, but the odds are twenty to one that it is not additive.
Q. Let me back up.
I want you to assume for a minute that you have a study on smoking asbestos interaction for lung cancer and the 95 percent
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confidence interval embraces the additive result, okay, do you understand what I am asking you?
A. Yes, you are saying it gets - it can be anywhere. If it's just in it, that means the chance at that is the appropriate

model - is one in 20.
Q. Actually, does it not in fact - it means that you cannot reject the additive model as equally as possible as any other value within that confidence interval, isn't that right?
MS. DIX: Objection.
A. Not at all. You cannot say that that model is equally appropriate to the multiplicative model if the difference between the multiplicative additive is such there is only a five percent chance that it could be additive.
Q. Okay, let me make sure I follow. Is it your testimony, sir, that if the additive model cannot be rejected by a scientific study, that is it falls within the 95 percent confidence level, that you can

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still draw the inference that it is more likely that a multiplicative model would apply?
A. It's certainly more likely than not and it's more likely than not with a very - to a very strong degree.
THE VIDEOGRAPHER: Going off the record it's 2:45 p.m.
(Recess taken.)
THE VIDEOGRAPHER: Back on the record continuing the deposition of Dr. Nicholson, it's 2:57 p.m.
BY MR. SCHROEDER:
Q. Dr. Nicholson, before the break we were talking about the studies, epidemiological studies, addressing lung cancer, smoking and asbestos; do you remember that?
A. Yes.
Q. The risk rates that are reported by Dr. Selikoff for interaction between smoking and asbestos for lung cancer are among the highest rates among those groups of studies that address the issue of interaction,

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are they not?
MS. DIX: Objection to the form of the question.
A. There are one of the largest in terms of exposure. There are included in groups studied factory workers in Great Britain, I think the exposures there are comparable in terms of intensity. I don't know how many of the factory workers are as long term as the insulators, but they are - it's a heavily -- insulators are a heavily exposed group, not the heaviest, but heavy.
Q. The rates are much higher than most of the other studies, at least as reported in the table by Ehren on the studies

that he looked at, aren't they and those are the rates for the Selikoff studies; isn't that a fair summary of the ratings?

MS. DIX: Objection to the question.

A. I don't know, let's see what the lung cancer rates are. RERI is the relative excess risk to interaction.

Q. It has over in the left column the

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rates for asbestos or rates for smoking, then the joint rate, do you see those?

MS. DIX: Objection.

Dr. Nicholson already testified he doesn't agree with Ehren.

Q. I'm asking about the data.

A. It has lung cancer risk ratios.

Q. Do you see the Hammon '79 study, Selikoff/Hammon '79?

A. I see Selikoff and the risk ratio for smoking of 8.7 and for smoking and asbestos exposure of 40.6.

Q. That 40.6 is much higher than the rates for most of the other studies on that table, right?

MS. DIX: Objection to the form of the question.

A. There is another vary of 35.5. There is Hammon of 53.2; from the factory, 31.9, from another study of Berries, then there are others with lower risk ratios.

Q. And the highest one is the Hammon group?

A. That's correct.

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Q. And that's fairly constant among the studies, is that the Hammon results, the Selikoff/Hammon results on lung cancer smoking interaction are at the highest end of most of the reported studies?

MS. DIX: Objection to the form of the question.

A. Well, they are at the top part of the range, but there are others that are very similar, like Selikoff is what 43 and some other one is 39, so there is four or five that are above 30 and you have six or eight that are below 30.

Locking --

Q. Go ahead.

A. No.

Q. Is it fair to say, Dr. Nicholson, that the studies addressing the relationship between asbestos, smoking or for lung cancer provide results that range from the additive to the multiplicative?

A. There are -- there are value -- I reviewed 16 articles, six suggested interaction between asbestos and smoking that

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was greater than directly multiplicative. Six indicated an interaction that it was directly multiplicative for suggested and interaction less than multiplicative, in one or two cases additive.

Q. My question is is it fair to say the studies that address lung cancer synergy for asbestos and smoking range it from the additive to, in this case, more than multiplicative?

A. The additive in number is very, very few in the studies reviewed, but there isn't - I do remember one study was additive.

Q. Have you rejected the additive studies?

A. Yes, as a general -- I didn't reject the study, I reject additive as being the interaction between cigarette smoking and asbestos for lung cancer based upon the total array of studies.

Q. Do you believe epidemiologically that a multiplicative relationship between asbestos and smoking for the risk of lung cancer applies equally, notwithstanding your

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asbestos exposure?

MS. DIX: Objection to the form of the question.

A. Yes, I do.

Q. So if somebody is exposed to less than the exposure of an insulator, you would still apply a multiplicative model to that?

A. Yes, most of these studies where the multiplicative studies are, they are.

Q. Are there any studies - let me - are the studies you would rely on for that proposition the ones contained in your report?

MS. DIX: Which proposition?

Q. That support the notion that a multiplicative model can be applied to any asbestos exposed person irrespective of their exposure?

A. They are in detail in the study of Vainio, which is referenced in the report with the results from that study there.

Q. Dr. Nicholson, I have handed you what's been marked as Exhibit 5, you have taken a look at that at the break, right?

A. Yes.

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Q. Those were documents produced to us pursuant to the subpoena; could you tell me what those report and reference the WN number at the bottom of each page when you describe each document?

A. In WN 6856, there is a graph of

asbestos-related mesothelioma cases that I calculated in a 1982 paper to be expected in the ensuing years.

It's one in which the cases in the United States rise to about 3,000 around - between 1995, 2000 and then tail off. There are points and two NCI programs that attempted to quantify mesothelioma deaths in the United States on the left of the graph.

The asbestos related cases that I calculated were the expected actual cases and that involved analysis of rates considering all material, autopsy, clinical and what material identified the mesothelioma death and then simply the death certificate deaths.

Q. Have you updated this chart since 1982?

A. No, because it happens to be right

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on the mark.

Q. Would you stand by this for the rates today?

A. Yes.

Q. Are these absolute number of cases or are these --

A. Yes with ballpark 2,800 cases of mesothelioma in the United States at this time, 28, 2,900. I might -- that's it.

Q. The next document should be WN 6877; is that correct?

Read me the next one.

A. I have got 6845.

Q. What is that one?

A. It's a graph of mesothelioma risk among insulation workers according to time from onset of exposure.

Q. Describe for us what this is telling us.

A. It's the risk of death in terms of cases per hundred thousand per year, to my recollection and it indicates that the risk of mesothelioma rises very, very steeply time from onset of exposure to about 50 years from

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onset of exposure. Thereafter it's uncertain.

There is a lower point, that point happens to be - I think it's made up of four cases. It's also uncertain, because any insulator that's lived to be 55 years from onset of exposure, probably had a very low exposure, he became a business agent right away and so it's unlikely to be comparable to the others and thus the best description of mesothelioma risk for 50 years of onset of exposure is one according to the third power of time minus ten years; cubic time line is ten years, times some other facts.

Q. Is this among an insulator cohort?

A. Yes.
Q. Is this among the 17,800?
A. Yes.
Q. The data derived are from --
A. Yes.
Q. For someone whose 40 years from onset as an insulator, are those people still alive today?
A. Some of them are.

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Q. And you would expect more cases of mesothelioma from that group in the future?
A. Yes. Right now the group would have a lesser risk, because they have not had an exposure of consequence since 1972, so the people dying now are among lesser exposed cohort. This is analysis of the mortality of insulators through 1976.
Q. And so if I were looking at this chart, just to make sure I understand it and I am 40 years from onset, my risk for mesothelioma has increased how many fold?
MR. KAZAN: Compared to what?
Q. Compared to when I started my job as an insulator.
A. Probably a thousand fold or thereabouts. I mean this is already starting at a risk much higher than background.
Q. Are you saying it would be even higher than a thousand fold?
A. Perhaps.
Q. What's the next one you have there?
A. I have more mesothelioma graphs.

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Q. Tell me what the number is on that one.
A. 6866.
Q. Is this a document that you prepared at one point in your career?
A. Yes.
Q. And tell me what it is.
A. It shows - in this case it's not risk of mesothelioma, it's risk of lung cancer in which I depict on the left an absolute risk of lung cancer.
Q. This is among the insulators again?
A. No, this is just a hypothetical group, it's schematic and not specific to a group. I assume that the lower line in the left is the underlying risk of lung cancer, which would be that of a population that does smoke cigarettes or has cigarette smokers among it and this indicates two exposures to asbestos that increase the risk of approximately four fold and so the line on the left depicts that four-fold increase taking place over 20 years and another one similar

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four-fold increase over a later 20 years and the relative risk for each of these is depicted in the upper right and it shows a relative risk that raises from one start of exposure to four and the excess risk in the bottom is showing which exposure beginning at age 20 raises modestly.

The exposure in excess risk from the exposure beginning at age 20 raises dramatically because the underlying risk is much greater other than over the years, so it indicates the --

MR. SCHROEDER: He needs to change the tape.

A. -- that the fact of asbestos at older age is more dramatic because of the underlying risk of lung cancer being greater. Of course this 20-year risk continues, it will also rise steeply in absolute terms after age 40.

THE VIDEOGRAPHER: We are going off the record. This is the end of tape two, it's 3:15 p.m.

(Recess taken.)

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THE VIDEOGRAPHER: Back on the record. This is the beginning of tape number 3, it's 3:16 p.m.

BY MR. SCHROEDER:

Q. Dr. Nicholson, was there anything else that you wanted to talk about on 6866?

A. No.

Q. What's your next one?

A. I have --

Q. That's 6865?

A. Yes.

Q. What is that, please, sir?

A. This is a graph of lung cancer risk among a factory population that manufactured amosite insulation during and after World War II.

The population had a high turnover, because the plant was extremely dusty and so the group under observation was one that worked a short number of years, one two or three and this is their mortality experience. This measures years of onset of first exposure.

Q. This is for lung cancer?

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A. For lung cancer. There was no evident increase in the first ten years; thereafter the lung cancer increased five times than expected - was manifest.

Q. And that was for three years or less exposure on average?

A. On average, yes, so you can get plants that are completely horrendous in their effects.

MS. DIX: This is amosite?

THE WITNESS: Amosite, pure amosite.

Q. Now we are at 6877; is that a document you created as well?

A. It looks like a slide I might have made for a lecture for medical students. I believe it's something I created.

Q. It's entitled Criteria for Evaluating Epidemiological Studies.

A. Yes.

Q. Are these meant to be - what are these things that are listed here?

A. Well, different criteria that one would use in evaluation; consistency, the

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studies of the same phenomena giving similar results.

The strength of the evidence is study a few - number of people, a large population where you have more accurate measures of increase.

We'll come to dose response relationships later.

Specificity, do the studies reflect a specific agent of concern or are they ambiguous in what the exposures are.

What are the time relationships of any manifestation of effect and we have gone into that a lot already.

What is the dose response relationship; is it that you see an increase in risk with increase in exposure; is it linear, is it something else.

Biological plausibility, the effect manifest one that might have readily understandable biological cause, it doesn't mean that we know the cause, but is it plausible. And what is the effect of removal from exposure, does the risk go away, does it

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continue or what are its -- what is the relationship of time from removal of exposure.

Q. Dr. Nicholson, you have listed here seven factors, is any one of these the most important or more important?

A. Well, I think time relationships and dose response relationships are particularly important, specificity is as well.

If you can't tell that the effect is due to an agent because of compounding exposures, you have trouble, but usually the specificity is fairly easily fulfilled and the others would be secondary to those.

Q. What is the last page which is

marked 6879?
A. This is a depiction of the world to my awareness, I believe it's the world production of asbestos varieties over time and it's depicted according to the three commercials principal, commercial varieties, chrysotile, amosite and crocidolite. It shows there is a modest use of crocidolite early in the 1950's through 1969 period of time and

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then it is largely not used.
A greater use of amosite beginning in 1945 and increasing and leveling off in 1979. The amosite mines in South Africa shut down some time in the '80s and there is no production after 1980 and the chrysotile production is the dominant one and it rises to a peak in the 1970 to 1975 period of time and in 1984 it decreased by 50 percent and there is no data after 1984.
Q. To what extent - did production of asbestos follow the same trends?
MS. DIX: Objection.
A. I think for chrysotile they probably increased similar, maybe not, because you get a mill running at capacity and you go with that.
There certainly was an increase over time and I believe the United States production would have been ceased either before or shortly after 1970, so our production would have decreased before the production decreased here.
Q. Do you know when the

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Johns Manville Company, the time period during which it manufactured asbestos in the United States?
A. I know that it was manufacturing thermal insulation until at least -- I believe, they were manufacturing thermal insulation with asbestos up until 1972 at which time the first OSHA regulation went into effect and the regulation requiring monitoring of the work place, recordkeeping and medical examinations for insulators led to contractor complaints and the manufacturers took asbestos out of thermal insulation and I think other uses of asbestos were vanishing at that same period of time, so some time in the early 1970s, I believe, Johns Manville would have gotten out of the asbestos business, but I don't know explicitly the years.
I know that asbestos use in the United States dropped dramatically in the early 1970s.
Q. When did the first study become published that to your satisfaction established the dangers of asbestos for

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insulators?
MS. DIX: Objection to the form of the question.
A. Well, the dangers of asbestos exposure were known in the turn of the century, shortly thereafter.
There is a report of the surviving member of a group of ten people working at a textile mill and dying from asbestos -- of asbestosis, all his comrades or I guess maybe it might have been a woman.
In any case, the co-workers were already deceased, but that was first published with the mention of asbestos disease over the ensuing years.
Asbestosis was noted in different reports, certainly in the 30s. I think Selikoff was the first to report on insulation workers, but well before his report it was clear that asbestos exposure caused harm and certainly asbestos workers were known to be exposed to heavy quantities of asbestos, so that's what we have.
Q. So if I read Exhibit 6879
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correctly, as applied to what you said about in terms of how U.S. production would mirror that, would it be fair to say that Johns Manville continued to produce asbestos for use in the United States beyond the time at which the risks were known, at least to the company and beyond the time that Dr. Selikoff himself reported on those risks?
MS. DIX: Objection to the form of the question. Mischaracterizes his testimony. Dr. Nicholson said he really wasn't sure exactly when Manville stopped manufacturing asbestos.
MR. KAZAN: Are you not distinguishing between this chart which summarizes the mining of raw asbestos fiber and the manufacturer of asbestos-containing products?
Your question assumes it's the same thing.
MR. SCHROEDER: What I am asking is --
Q. Isn't it - is it your testimony then, as I understand it, that well beyond the
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point that the risks and dangers of asbestos were known to companies like Johns Manville, Johns Manville continued to manufacture and put into the marketplace asbestos-containing products?
MS. DIX: Objection to the form of

the question.
A. Manville did produce asbestos products after Selikoff's 1964 study and after other studies indicated asbestos exposure and lung disease, asbestosis.
I can't tell you how much longer afterwards, but I believe that in the United States they ceased production - I know they ceased production of thermal insulation of asbestos in 1962, everybody did.
What has taken place abroad at Manville, I don't know.
Q. I'm worried about the United States.
A. I told you what I know. I know they were producing asbestos products in '69, but I don't know - I can't say more or I said what I know thereafter.

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Q. We talked earlier about threshold and I want to ask you a question about mesothelioma.
Is there any safe threshold for mesothelioma?
A. Not that I know of.
Q. And that's OSHA's position as well?
A. Yes, it is, as it is in lung cancer, but the evidence of low level exposure mesothelioma is stronger than that of lung cancer, because you don't have the ambiguity in lung cancer of other factors playing a role.
Q. Is the latency period for mesothelioma longer or shorter than the latency period - than lung cancer generally as a rule?
A. Generally it's longer. It's hard to say. You see from the graph that was shown of Selikoff's that it was in fact - he had no cases of mesothelioma until 15 years from onset of exposure and that graph reflects 179 cases, so cases have been reported for shorter

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than ten years, but it's very, very rare for it to occur for 20 years and then it rises steeply and you get cases coming strongly after 30 years.
Q. If I were an insulator and I want you to assume two scenarios, one is I am an insulator and I develop lung cancer, okay and let's assume my lung cancer - that the cause is unknown, okay; is that a scenario that you - an assumption you would make or would you assume an insulator who gets lung cancer would most likely have lung cancer because of asbestos?
A. Or because of smoking. Those two - I would immediately want to know his

history of smoking and of asbestos exposure.
Q. I want you to assume for a minute there is an insulator who develops lung cancer and further assume that the claim is made that it's related to smoking, okay?
A. Okay.
Q. If that insulator did not develop lung cancer and be fortunate enough not to have gotten lung cancer, would they still be

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at risk for getting mesothelioma?
A. Yes, according to whatever asbestos exposure was and that's a high risk for a person working steadily, continuously as an insulator.
Q. If you would take a look at your report, Dr. Nicholson, and turn to page 29, table 23, do you see the table there that summarizes your values for pleural abnormalities?
A. Yes.
Q. That's what table 23 is meant to do?
A. Yes.
Q. And for pleural fibrosis and pleural plaques, would you agree with me that the lower confidence levels for both of those go below the background level of 1.0?
A. Yes, that's correct.
Q. Would you further agree that drawing from that chart that the conclusion one would reach is that you cannot say that pleural fibrosis and plaques are related to smoking to any statistically significant

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degree?
MS. DIX: Objection to the form of the question.
A. You can say that pleural abnormalities are --
Q. Let's start with fibrosis and plaques.
MS. DIX: Let him finish the answer.
Q. I want an answer. My question is -- I don't want - you can answer the rest of it, but I want an answer to my question first.
For pleural fibrosis according to your table 23, one cannot say that smoking has a relationship to pleural fibrosis that is statistically significant?
MS. DIX: Objection.
A. You can say that it does at very close to the 95 percent confidence level, so the likelihood of it having a relationship is extremely high and it's an arbitrary - at the arbitrary level of 95 percent it doesn't quite make that, it comes very close at the

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90 percent level; it certainly does have a relationship.
Q. At the 95 percent level which is - is that what you have reported here, the 95?
A. Yes.
Q. At the 95 percent level it is not statistically significant, is it?
MS. DIX: Objection. It's been asked and answered already.
Q. It's not at the 95 percent level, is it?
A. It's one percent off.
Q. I understand, so the answer would be?
A. That still means the chances of nineteen to one that it does have a relationship.
Q. Is it or is it not statistically significant at the 95 percent level?
MS. DIX: Objection, asked and answered.
A. It is close to being statistically significant, but is not quite.
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Q. And pleural plaques, are they or are they not statistically significant in the relationship to smoking at the 95 percent confidence level according to table 23?
MS. DIX: Objection.
A. They are also not quite, but the likelihood is very high that they are and certainly that one or the other of them is overwhelmingly likely to be very high likelihood.
Q. What do you mean one or the other; which one and which other?
A. It's more likely fibrosis than plaques, but if you consider a pleural abnormality, whether it be fibrosis, plaques, the probability of that occurring in this population with the ratio of the smoking to nonsmoking gives you a ratio of prevalence of 1.28 with a lower confidence level being 1.14, so that's well above one and thus classic pleural abnormalities either fibrosis, plaques, have a smoking effect in their development.
It's certainly less than
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parenchymal fibrosis and that's clearly stated.
Q. If I were considering simply pleural plaques, we would use that bottom line of pleural plaques, right?
MS. DIX: Considering pleural

plaques for what?
Q. Determining whether there is a statistically significant relationship.
MS. DIX: Objection to your question.
A. Well, from the data that's on pleural plaques, the chances are probably one in ten.
Q. My question simply, Doctor, if I want to -- if I am a trust and I say I am not paying fibrosis, I am not paying anything except pleural plaques, if you have a pleural plaques talk to me, if you don't, don't bring a claim, I would be looking then at whatever studies comprised your numbers that are on the line that say pleural plaques; is that correct?
MS. DIX: Objection to the form of

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the question.
A. It's also not reality, that's not what they are doing.
Q. I'm sorry?
A. That's not reality, that is the trust is paying more than pleural plaques.
Q. Have you looked at the claims files?
A. No, I haven't looked at the claims files.
MS. DIX: Which claims files?
Q. The files for the claim that's in the trust.
A. No, I haven't looked at any such files.
Q. Do you know how many there are?
MS. DIX: Objection. He just testified he hasn't looked at them.
A. No, I was told, but I don't know.
Q. To the extent there are claims solely for pleural plaques, then if we wanted to know what the prevalence is according to your testimony, we would go for the line of pleural plaques, would we not?

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MS. DIX: Objection to the form of the question.
A. At this point, that's what you would go with. One could also maybe do another review of the literature to see if there are more data on it in fact that would be - if it became crucial, if this number became particularly crucial, that would be an appropriate thing for someone to do.
Q. Would you agree, Dr. Nicholson, that the majority view in the literature is that smoking is not statistically associated with pleural abnormalities?
MS. DIX: Objection to the form of the question.

A. Well, I would agree that that might be the case at the 95 percent level. I did not make it statistically significant at that level.
Q. When we talked about the asbestosis prevalence studies, we talked about your analysis of a smoking versus nonsmoking dichotomy, correct?
A. Yes.

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Q. And your opinion in your report is that 50 percent of the cases of parenchymal abnormalities are or occur because of smoking, is that your testimony?
A. Say that again.
Q. Okay, your opinion, there are 50 percent more cases of parenchymal abnormality among smokers, is that a fair statement of your opinion?
A. Yes at the lower level and at higher levels of asbestos exposure that would be the case in terms of higher levels of X-ray abnormality.
MS. DIX: Perhaps you might want to look at your report.
MR. SCHROEDER: No.
Q. So the relative risk you adopt for the increase prevalence of parenchymal abnormalities among asbestos workers who smoke is 2.0; is that right?
A. Yes and that came from a review of a table. It came from analyses of studies under - on the table that I was looking at -- the table made up with different sets of

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information, so it throughout -- it came from Cox' regression analysis --
Q. You are welcome to complete your answer, but I was going to move to a related point and I know what's in your report. My point was not that one. I am trying to get to a related point. If you want to continue...
A. Go ahead.
Q. Is it fair to say among the whole group of people with parenchymal abnormalities, half of the parenchymal abnormalities are caused solely by asbestos?
A. Not necessarily. You are now going to a biological phenomena. So there can be joint action across the board and that result is that what evolves is that there is twice as many abnormalities as would be the case had they not smoked.
You can't tell on an individual how to apportion causality, you can only do so on a statistical basis for the entire group and one finds roughly that the spectrum of abnormality seen is the result equally of exposure to asbestos and cigarette smoking.

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Q. Is it inherent in a statement that there is a 50 percent increase prevalence that the other 50 percent occurred not because of smoking, but because of some other factors including asbestos?
MS. DIX: Objection to the form of the question.
MR. KAZAN: We have now gone from 50 percent of the total to 50 percent increase and that's exactly not what his opinion is and I don't know whether it's just confusing terminology or perhaps intentional, but I think it's getting very confusing.
A. It's double; it's a hundred percent increase, it's double.
Q. So if it's doubled, are you saying that half of them are not - have no relationship to smoking?
A. No, I am not. I am saying I don't know if it's half.
Q. Not half of the increase, half of the total?
A. Of the total. I am not saying
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there is a half out there in which smoking played no role whatsoever and it played the dominant role in the other. I am just indicating that it is the combined effect of the totality of the group is such that there is a doubling of the number of cases of parenchymal abnormalities.
It could be that it is a joint effect on all of them so they are all raised a bit, but the net result of the raise is only half, that there is twice as many abnormalities that would be the case with asbestos exposure alone.
Q. In the area of lung cancer risk, it was your view, I believe, that you need to allocate the excess risk between asbestos and smoking, right?
A. That you can do.
Q. In the area of asbestos?
MS. DIX: Let him finish.
A. You can do so here, but with less certainty, because we don't fully understand the biological mechanism. We have better information on that -- more information on
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lung cancer that would suggest that the Chase Kotin model is a good one to use for allocation.
Q. What I want to ask then is in the area of asbestosis, wouldn't you want some model to adopt there to divide up the excess

risk between smoking and asbestos for pleural abnormalities?

MS. DIX: Objection. Again, you keep using the word asbestosis and Dr. Nicholson is using parenchymal abnormalities.

Q. Same question.

In the area of parenchymal abnormalities, wouldn't you want some model to divide up the excess risk there?

A. That would be nice to have, but it isn't necessary to have to evaluate what would be the smoking contribution to the totality of the disease and to allocate costs related to that contribution, whether that contribution be an amount on everybody's X-ray or dramatic amounts on certain people.

You are looking at a large

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population and you look at the difference between that and the equivalent population to see the differences, manifestations of the fact. Whatever compensation is appropriate for that effect, the -- does not depend upon the model of the interaction.

Q. Why not?

A. Because I can't imagine there being any different payment plan in this case to the trust, because we are not dealing with individuals, whether the smoking effect was broad across the group or as you are trying to put it only affected half the group and the other half is totally unaffected.

I don't believe that's the case at all. I believe there is a smoking effect on the X-rays of everyone, but that it may not or some people lead to a change in X-ray, but the net result in the population is that it produces twice as many abnormal X-rays.

Q. Would you agree that we presently -- science doesn't know what biological mechanism, if any, there is for an increase in parenchymal abnormalities among

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smoking asbestos workers?

MS. DIX: Objection to the form of the question.

A. We don't know exactly, just as we don't know the asbestos effect for lung cancer. There have been suggestions of what the effect is that have certain reasonableness about it and we may know more of it in the future, but right now I would certainly say that we do not have a definitive answer as to mechanism.

Q. Do you consider yourself to be an expert in the area of biological mechanism?

A. No.

Q. Would you defer to -- do you know

Dr. Arnold Brody?
A. Yes.
Q. Would you --
A. Sometimes I disagree with him on that, but he certainly is knowledgeable and whatever his opinion is it has to be listened to, because it's put forth with knowledge and skill, but he also may not be correct. I'm indicating that it's possible, but you listen

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to him.
Q. He would be more informative in that area than you would be?
A. Yes, indeed.
Q. Now, putting aside the issue of compensation that you referred to just a minute ago on compensation schemes, from a purely scientific point of view, shouldn't the excess risk of parenchymal abnormalities found under your prevalence analysis be allocated between smoking and asbestosis?
MS. DIX: Objection to the form.
A. No.
Q. You are assigning it all to smoking?
A. We are assigning -- I'm assigning the excess to smoking, because that's the extra number of cases that occur because the smoking was present.
I am not saying that smoking did not play a role jointly in any individual's risk, because it could.
I'm indicating smoking could play a role across the board. The net effect of

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that is to produce twice as many cases of abnormality than would have been the case had smoking not been present, so this doubling of the prevalence is a smoking effect added to what is already asbestos effect.
Q. Do you then regard the 50 percent increase number of parenchymal abnormalities in your prevalence analysis that you find with smoking, do you then regard them to be not related to asbestos?
A. No.
MS. DIX: Objection to the form of the question. Mischaracterizes his testimony.
A. If asbestos is playing a role in those as well -- if you want to allocate the asbestos component and a cigarette component to these new cases, fine. You also then must allocate a smoking component and asbestos component to the other cases, because they are two -- they are also affected by cigarette smoking, so you can do it one way or the other.
The net result is that you detail

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all these mechanisms, which we don't, is that you will come up with the same number of cases attributable to cigarette smoking as you do simply looking at the excess number, but you can't be unfair and say these new cases we are going to look at the cause to be the result of both exposures and not do the same with the ones, the other ones even though you don't know which is which.
Q. Son under your analysis looking at the group as a whole you would assign the 50 percent increase number to tobacco, do I understand that right?
MS. DIX: Objection to the form of the question.
A. I would assign all of the excess that is seen, the doubling of the number of X-ray abnormalities in a smoking population compared to a nonsmoking. I would expect that X-ray to be due to the cigarette smoking which is the difference between the smoking group and the nonsmoking group.
Q. I want you to assume for a minute, Dr. Nicholson, that in that group that you
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have just assigned all to smoking, that none of them --
A. I didn't assign any group all to smoking. Oh, I said -- for the population you have to attribute to smoking the number of the excess within the group itself all are affected by smoking and some of the group are affected to the degree that the X-ray reading is changed by one or two units, but any way you look at it there is no way you can get around the simple fact that the smoking group has doubled the abnormalities.
Q. Now, I wanted you to assume that none of those persons smoked in your group where you have a doubling effect, okay?
A. Well, if none of them smoked then none of them - the exposed group smoked, then you don't have any increase in X-ray prevalence.
Q. In absolute numbers though many of the people who didn't smoke will still present with asbestosis at some point in their life if they live long enough, won't they?
A. Indeed.
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Q. If you assume that there was no smoking, okay?
A. Then there is no smoking effect and we can all go home.
Q. Well, it's a little more complicated than that, I think, isn't it?

A. No. If there is no smoking there is no problem with smoking.
Q. The trust has decided it's going to pay anybody who presents with a claim for asbestosis no matter when you present, as long as you file your claim before the year 2049?
MS. DIX: Objection to the statement.
Q. Do you understand that?
A. Yes.
MS. DIX: Is that a question?
Q. Understanding that the trust will pay claims filed up until the year 2049, my question is in a world with no smoking, a lot of these people who you might say their asbestosis is attributable to smoking would still file a claim to asbestosis, it will just come later down the road if they are fortunate

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to live long enough?
MS. DIX: Objection to the form of the question.
A. We are not in -- the hypothetical is not real.
Q. I want you to assume my hypothetical.
A. If nobody has ever smoked then there is no smoking effect in the asbestosis, it's as simple as that.
MS. DIX: Objection.
Q. I understand.
A. Everybody, not everybody - there has been a large component, 80 percent of people that filed for asbestosis have smoked.
Q. Right?
A. So smoking plays a role.
Q. But for the purposes of the trust, they will pay your claim as long as you file before the year 2049, you understand that?
A. Yes.
Q. And if that's true, then in a world with no smoking, a lot of the people who were developing parenchymal abnormalities

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because of a smoking effect under your report would still develop a parenchymal abnormality without a smoking contribution that would just develop later in time?
MS. DIX: Objection, hypothetical.
A. Some of them would, some of them might not. The number that would be similar to that would be the number that you would estimate on a basis of a no smoking effect.
Right now we looked at the smoking effect in comparing smokers with nonsmokers and you find this twofold difference. If you get in a future 30 years from now when nobody has ever smoked, then be you can be dealing

with a pure nonsmoking group, but we are far from that at this point in time. You are looking at one with a high smoking prevalence.

Q. I am asking you to assume. I understand you are saying that's not reality.

A. If you get 40 years down the line and you have such a group then you don't have too much a smoking effect and as you move through that group you can make adjustments

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over time, but now we are dealing with what was out there in the past.

Q. If you have - let's deal with some concrete numbers in an example.

If I have a hundred people who present with asbestosis and let's call it parenchymal abnormalities, since that's the definition you use in your report, okay and you apply your prevalence analysis to the hundred with parenchymal abnormalities, tell me first of all how many of the hundred would you say are presenting claims because of a smoking effect; can you tell me that?

MS. DIX: Objection.

A. No, I don't know.

MS. DIX: Objection to the form of the question. What do you mean presented claims?

A. I have no idea what any of the exposures are, so I don't know what the likelihood of presenting is, even if they didn't smoke that would -- that's a prerequisite before I can answer your question.

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Q. Do you understand in this case Dr. Harris has presented a model for a world with less smoking?

MS. DIX: Objection to the form of the question.

A. I have heard that. I know he is certainly treating issues of smoking and I don't know what his - what he has done and that's --

Q. Have you read his reports?

A. No, I have not.

I mean I talked with him briefly, but I have not discussed in detail what he is calculating and what he is doing.

Q. What discussions have you had with Dr. Harris?

MS. DIX: With Dr. Harris?

MR. SCHROEDER: Yes.

MS. DIX: You are inquiring into areas now which were past the submission of Dr. Nicholson's report.

MR. SCHROEDER: Dr. Harris is a testifier in the case who says he relies on Dr. Nicholson's report.

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MS. DIX: What is your exact question?
MR. SCHROEDER: If Dr. Harris is a testifier who is relying on Dr. Nicholson's report and they had discussions about that, I think it's discoverable information.
MS. DIX: You are asking about their conversations to the extent they had any?
MR. SCHROEDER: First I am asking whether they had discussions, he said yes.
A. We had discussions about what information we had on smoking in this group of 2907. I told him, he said that sounds good, send me stuff, I did.
Q. Did you all discuss the substance of what is in the group of 2907?
A. We discussed - I discussed what the group was and the information we had on it, particularly with respect to cigarette smoking, which is detailed for each person.
Q. Have you met with Dr. Harris?
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A. No.
Q. How many different times have you talked with him?
A. On the phone, three, four.
Q. How long were your discussions?
A. A couple were fairly long, 15 minutes to a half an hour; he is a talkative person, I was mostly listening to him.
Q. When was the nature of your discussion about, the information in the 2907?
A. That's correct.
Q. What did he want to know about that?
A. What we knew about the group, what we were doing with it. I described in detail the - basically the non-clinical information that we had on the group. He went -- he didn't care that much about the examination data.
Q. I'm sorry?
A. He did not care that much about the examination data; we did not talk about that.
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Q. The examination data being the clinical data on the individuals?
A. Right, whatever a physician looks at, say ah and that sort of stuff.
Q. How about medical reports, was he interested in those?

A. No, I mean not beyond much -- he has had I am sure other discussions with Dr. Levin on this, but I was the first one he spoke with.
Q. When were your discussions with Dr. Harris; when did they start, if you recall?
A. A month ago, maybe a month and-a-half.
Q. Can you tell me, Dr. Nicholson, based on your knowledge of the epidemiology dealing with the issue of smoking and parenchymal abnormalities whether in a world without smoking whether there would have been more claimants to the Manville trust or fewer claimants to the Manville trust for parenchymal abnormality?
MS. DIX: Objection to the form of

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the question.
A. I believe there would be fewer in a world without cigarette smoking.
Q. What do you base that on?
A. Because of the evidence there would be fewer parenchymal abnormalities present in the population.
Q. Over a period of time as you progress from time since onset, even if you don't smoke, your risk of getting asbestosis increases, does it not?
A. That's correct. Correct. If you are continuing exposure it increases with increasing exposure. It also increases with time absent increasing exposure.
Q. And so would you agree with that even among the smokers in a population of asbestos exposed individuals if they don't smoke?
A. Say that again.
Q. You have smokers - you have a group of smokers in an asbestos exposed population, okay, call them insulators.
A. Yes.

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Q. Let's assume they smoke for 20 years.
A. Right.
Q. I want you to assume the same group, but those people don't smoke.
A. All right, so they never smoked.
Q. They never smoked, right.
A. Yes.
Q. At the end of the day if they are all fortunate to live long enough, you are going to have about the same number of claims for parenchymal abnormalities if they are all fortunate to live long enough just by virtue of the passage of time, are you not?
MS. DIX: Objection to the form of

the question.
A. There would be more claims in the smoking group than in the nonsmoking group dealing with a 20 year period of time.
You consider 40 year insulators over time, they are all going to get some abnormalities and there will be a difference in severity for cigarette smoking, but you will have fairly similar, but still a greater

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number of filings by the smokers, but the difference will be greater in earlier years.
Q. I understand your opinion is the difference to be greater in the earlier years. My question is in the later years doesn't it eventually catch up because of the passage of time in the risk for asbestosis?
MS. DIX: Objection to the form of the question.
A. It may nearly catch up, but I don't know it quite well. We are looking at a very unique sub group, one that has very high and continued exposure for 30 or 40 years.
The majority of claimants, I believe, the exposures are much lower and the difference between smokers and nonsmokers more distinct.
Q. Have you done any analysis to determine in a world without smoking how much more or fewer claimants there would be for parenchymal abnormality?
A. I haven't had any claimant data, but simply put, the chances of developing an abnormality are half as much if you don't

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smoke than if you do, so one can hypothesize a particular smoking level in a population and calculate according to that percentage the relative number of filings or develop cases of parenchymal fibrosis that would evolve.
As you go from a group that was say 100 percent smoking to a group with no smoking, the percentage of filings that are increased because of the group smoking would decrease according to the number of never smokers in a population, so that can easily be done and it's a very simple test.
It would be would be one that would be direct -- the filings would be directly -- the excess filings would be directly proportional to the percentage of smokers.
Q. But that analysis doesn't take into account the passage of time, does it and the effect passage of time has on whether you eventually present with a parenchymal abnormality?
A. Well, it depends on - it does take into account the passage of time as

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smokers die off in this hypothetical group that you have evolving into a group of never smokers. Right now actuality will not have a group of never smokers for 20 or 30 years.
Q. And the smokers who die off will then be living in the other group, wouldn't they, they would be alive in a world with no smoking?
A. The smokers that die off.
MS. DIX: Objection.
A. That die off will be dead.
Q. In a world with no smoking they may not have died, right?
A. A comparable person may end up dying, that is the case. There will be more people alive among the nonsmoking group as time progresses and you can have a group longevity as it were and calculate what the mortality rate with time would be given standard models.
Q. With more alive, you could actually have more cases of parenchymal abnormality being filed couldn't you?
MS. DIX: Objection to the form of
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the question.
A. The data would be such that that would not be the case given equal criteria for the filing. A doubling of cases is not going to be matched by a slight increase in longevity.
Q. At a one over zero compensation threshold, if you would assume a one over zero compensation threshold in a world with no smoking, at that threshold you could actually have more people filing than at a higher threshold of two over two or three over three?
MS. DIX: Objection to the form of
the question.
A. My previous answer applies directly to that. You are not going to get a doubling by a slight increase in the cases developing.
Q. You testified earlier you were involved in the development of green sheets?
A. Yes.
Q. Did you start the green sheets; was that your project?
A. Yes, that was one of my jobs.
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Q. When did you start the green sheets?
A. They started in the first quarter of 1969. I don't think I wrote the first one, Selikoff did that all on hand.
Q. The green sheets were warning

sheets to asbestos workers about the risks of asbestos?
A. In part. They were also discussions of mechanisms that were being developed and available to reduce your exposures and they dealt with reports of meetings of asbestos workers who Selikoff gave the talks and they asked questions, so there is a question and answer session so the question and answers would be written up.
I did the best I could to put something in that would be relevant and interesting and I don't think it was read anyway.
Q. Why do you say that?
A. I know a lot of people said we never read that.
Q. Who said that?

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A. Some of the insulators at times.
Q. Who did you send them to?
A. Every insulator. It was an insert into their journal.
Q. Did you submit them to other unions as well?
A. Yes.
Q. What other unions did you send them to?
A. I don't remember.
Q. To the best of your knowledge, were all the unions that would have been unions with workers that may have been exposed to asbestos sent copies of green sheets?
A. I think that was probably the case as far as we can determine. We had a mailing list of industrial hygienists of union leaders, of government people that we felt might have some interest, whether they did or did not, I can't say, but it was an extensive mailing list outside the regular union members.
Q. Is the mailing list still maintained at Mount Sinai?

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A. No.
Q. Who would have the mailing list today?
A. I have no idea.
Q. How often were the green sheets published?
A. Quarterly.
Q. Do you know how many were printed every time they were published?
A. Well, the membership tended to be about 18,000. We sent it to the -- we didn't get involved in it.
Q. You sent the original and it went off?
A. Yes, you send the original to the

journal printer, he printed it, put it in the journal and he printed for us a batch to be sent and I don't know whether he had a mailing list and they sent them out. I don't remember how it got out to the other groups, but they were certainly sent to people like those that I just mentioned.

Q. Would it be fair to say they were widely distributed among folks who were

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exposed to asbestos?

MS. DIX: Objection to the form of the question, lacks of foundation.

A. Well, they were widely distributed among the insulators, a group that was one of the heaviest exposed groups, but we didn't -- the number at that time it was 17, 18,000. There are 20 million people over time that have been exposed, for example, five million shipyard workers in World War II and ten million construction workers thereafter.

Such workers did not get them individually, some of their leaders may have. We certainly tried to get them to principal union members of construction unions and whether they in turn put a bit in their own journal, I don't know.

Q. Mount Sinai felt that it was important to warn people exposed to asbestos of the potential health effects?

MS. DIX: Objection to the form of the question. Mount Sinai, Dr. Nicholson or both?

A. It's important to have people's

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exposure to asbestos reduced to the greatest amount possible and eliminate it if so.

To the extent that warnings play a role, we gave them. In fact I think actions by others other than the workers could play a greater importance in particular contractors using saws that were properly ventilated and doing and having industrial hygiene measures in place, so we were going after those people as well.

THE VIDEOGRAPHER: Going off the record, 4:20 p.m.

(Recess taken.)

THE VIDEOGRAPHER: Back on the record continuing the deposition of Dr. Nicholson, it's 4:26 p.m.

BY MR. SCHROEDER:

Q. Dr. Nicholson, we were talking about the green sheets before we took a break; how long were the green sheets published through Mount Sinai?

A. I believe it was seven years.

Q. They started in what year?

A. They started in the first quarter

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of 1969 and you have the set, I think it's seven years.
Q. Why did you stop?
A. It was getting repetitive, we did not have an industrial hygiene program at that time and the insulators were no longer using asbestos in their products and certainly had occasion during removal, but we said I think all we could say.
Q. The green sheets addressed the issue of smoking risk as well?
A. Yes, it did. I believe the first article specific to smoking was in the third issue.
Q. How often did the green sheets cover smoking risk?
A. I don't know, I didn't count, but it would have done so several times and it may have made mention in different articles where that was it - was not the main focus, because in his interaction with insulators, Selikoff made major emphasis on smoking cessation, because it really did have an effect.
You can't do anything about the
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asbestos exposure risk, but you can about the smoking risk and correspondingly the combined risk.
Q. Did you speak about the issue of the risk of asbestos to workers around the country?
A. Yes.
Q. Tell me about what you did in that regard.
A. I did a limited amount compared to Selikoff. Anytime he was in a city and there was the possibility of giving - not anytime, but many times he was traveling through the West Coast or some other city and there was an opportunity to speak before a union meeting, he would do so.
I did so occasionally when it was - may have been a meeting and the union asked for somebody from us and he couldn't do it and I did it. I spoke in meetings of other unions as well, particularly the oil, chemical, anatomic, workers union that had a very strong health department, so to speak.
Q. Your speech at the union meetings
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would have started some time after 1969?
A. Yes, indeed.
Q. When did they start, do you remember?
A. Not exactly, but it would have been early, not that long, maybe 1970, '71.

Q. Did you travel with Dr. Selikoff?
A. Sometimes, depending on what the circumstances were. If I had something to do, for example, getting an examination site set up, we might go together. I certainly didn't go along just to listen to him speak.
Q. Did you ever give congressional testimony?
A. Yes.
Q. On this issue?
A. Yes.
Q. Tell me when you did this.
MS. DIX: Issue of what?
Q. On the issue of asbestos.
A. It ranged from the 1971 period until the 1980s and I did locate -- I have located recently testimony maybe four or five times for four or five instances.

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Q. So you testified four or five different times; is that right?
A. I believe so.
Q. Is that before the U.S. Congress?
A. Yes.
Q. And that's from the period?
A. Congressional committees, not the full congress.
Q. I understand. Those would have been committees dealing with issues relating to risk for disease from asbestos?
A. Yes, it would have been and it would have been over the time I indicated, say 1970 through early 1980s, thereafter. The asbestos issue was better known and much less a concern to congressional committees.
Q. Did your testimony deal with the effects of smoking?
A. Yes, that would have given -- I would have given the -- unless I was directed to be very specific in something or general lecture, I would particularly describe Selikoff's data as it was available from the 17,800.

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Q. Dr. Selikoff's study dealing with the issue of synergy with smoking and asbestos was published when, do you recall?
A. Maybe it was 1979, but he heads some limited data and spoke of it in 196 -- in an article in 1968, but the major study began in 1967 and there may have been some results earlier than the ten-year follow-up, but I think the classic five, 10, 50 analysis from 1979.
Q. Do you know when Dr. Selikoff then began warning asbestos exposed people about the synergistic effect that he was finding?
A. He was warning them very early. I mean he saw a synergistic effect and wrote of

it in 1968, so he was in speeches to union members, he would emphasize the likelihood of an interaction of cigarettes and smoking that went beyond the additive effects of each.

Q. Do you know how much after 1968 he began to warn about that?

MS. DIX: Objection, misstates prior testimony.

A. I believe he would have been

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warning workers certainly before 1970.

Q. Is it your understanding somewhere between '68 and '70 is when he would begin warning workers?

MS. DIX: Objection to the form of the question.

A. I believe he would, but I can't -- I don't recall such an event, but it would be characteristic of him to do so.

Q. When do you first recall Dr. Selikoff having warned about the issue of a potential synergy?

A. I don't have a recollection of that, I mean of the event. It's so much a part of what he does, that the first time is not one that I would remember.

Q. How many times over the years, Dr. Nicholson, have you made speeches or addresses to any kind of group who would have been exposed to asbestos, whether a union or not about the issues of the risks of asbestos and smoking?

A. I guess it would be two or three times a year from the early 70s to the

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mid-80s.

Q. Why did you quit?

MS. DIX: Objection to the form of the question.

A. The exposures to asbestos are considerably reduced so the number of groups requiring such talks has changed dramatically and the need for such talks was absent.

Q. So Mount Sinai sent out green sheets and you and Dr. Selikoff made presentations to various groups including unions about the risks of asbestos; what did you or Dr. Selikoff or anyone at Mount Sinai do to get the word out about asbestos and testifying at congressional meetings?

A. Presentations at scientific meetings and publications and the peer review literature and getting -- got involved at different times with the preparation of monographs, a pamphlet or volume, dedicated to asbestos health effects.

One was with the EPA, one was with Consumer Products Safety Commission, one was - I don't remember, but one or two

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others.
Q. How many people at Mount Sinai were engaged in the effort to investigate the health risks of asbestos?
A. In the 70s --
Q. Approximately.
A. A dozen professionals and then there was support staff. Maybe less than a dozen.
Q. Who at Mount Sinai helped select the persons from the American Cancer Society CPS-1 group to use as a comparison for the Selikoff cohort of 17,800 for the lung cancer studies?
MS. DIX: Objection, lack of foundation.
A. That would be Syler Hammon, Selikoff worked with him from the very beginning, very quickly and he provided the statistical support for Selikoff's epi studies.
Q. Did you work with Dr. Selikoff at looking at any quit rates of smoking for --
A. No.
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Q. You did not?
A. That's correct.
Q. Who would have; who did work with Dr. Selikoff on looking at quit rates of smoking?
MS. DIX: Objection, lack of foundation.
Q. Let's lay a foundation.
Are you aware whether Dr. Selikoff looked at quit rates for smoking?
A. He didn't do -- he obtained data on that, he did not publish a paper on it to my recollection. The most extensive data we have that I know of is from the 2907.
Q. Have you looked at the quit rates of the 2907, the smoking quit rates?
A. On a statistical basis, no.
Q. Do you know if anybody has?
A. Yes, Dr. Harris is working on -- worked on that and cooperating with us in its - it's an ongoing activity. Possibility exists that Selikoff with Hammon may have done something looking at the difference between insulators versus general population, but I am
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unaware of it.
Q. Are you aware of any data apart from what Dr. Harris is trying to do that addresses the quit rates of insulators, the smoking quit rates of insulators as compared to the general population?

A. No, I don't know of such activity.
Q. Have you had any discussions with Dr. Rubinowitz in this case?
MS. DIX: I am going to object. Dr. Rubinowitz is not a testifying expert in this matter and any such discussions would be discussions that are between consulting experts retained by the trust. If you want to be specific...
MR. SCHROEDER: I am only asking if he had the discussions, I don't know if there is a privilege issue here under your analysis.
Q. Have you had any discussions with Dr. Rubinowitz? You can answer yes or no whether you had any discussions with Dr. Francine Rubinowitz.

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A. Yes, I have in the distant past prior to the publication - prior to submission of this report we are speaking of.
Q. Your discussions were prior to your report?
A. Yes.
Q. I think those are fair game.
What did you talk with Dr. Rubinowitz about?
A. Well, we were joint meetings in Miss Dix' office and we described the progress we were making on our respective activities and I had brief conversations with Dr. Rubinowitz about what I was doing and briefly about the results. They were very limited, the conversation.
Q. What was the nature of the discussion?
A. I guess simply that I was finding a multiplicative interaction for lung cancer that could be - that was clearly established and that there was this doubling of incidents -- doubling of prevalence of abnormal X-rays among smokers compared to

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nonsmokers and a lesser effect -- we didn't talk about Pearl, but I believe I would have mentioned the parenchymal fibrosis information that I obtained, but it's a long time ago, I don't remember exactly.
Q. Who else was there from the expert point of view?
A. Probably Tom Florence.
Q. Have you met with Dr. Smith, Allen Smith?
A. Yes.
MS. DIX: Again, in connection with this report; is that correct, Tom?
A. I don't believe it was in connection with this case at all.

Q. It was in connection with something else?
A. I believe so.
Q. What was it in connection with?
A. I don't remember.
Q. How long ago?
A. It could have been in connection with this case, but I'm not sure.
Q. You don't recall when it was?

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A. It was some time a year ago.
Q. What was the purpose of that meeting?
A. I don't remember.
Q. Fair enough.
Have you read Dr. Florence's report in this case?
A. Not in detail. I glanced at it, parts of it.
Q. Did you --
MS. DIX: You are referring to Dr. Florence's September 21 --
MR. SCHROEDER: Any of his reports.
MS. DIX: Ask the question.
Q. Which one did you look at, Doctor?
A. I looked briefly at the introduction and scanned the report to be produced on September 1 of '99.
Q. Did you read it in substance to determine whether you could form any opinions about what he was doing?
A. No.
Q. If I were to make a statement to

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asbestos workers that unless they smoked even heavy exposures of asbestos over a prolonged period of time would not increase their risk for lung cancer over that of the general population, would that be true?
MS. DIX: Objection to the form of the question.
A. Repeat that question.
Q. Sure.
If I were to tell asbestos workers that unless they smoked even heavy exposures to asbestos over a prolonged period of time would not increase their risk of lung cancer over that of the general population of a known exposed persons, would that be true?
MS. DIX: Same objection.
A. That would not be true.
Q. Why?
A. Because there is an increased risk among asbestos workers who don't smoke.
Q. At what point in time based on your review of the epidemiology did that conclusion become evident?
MS. DIX: Objection to the form of

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the question.
A. Certainly by 1979 I think it was likely to be evident somewhere earlier and I can't site an instance.
Q. Can you tell me approximately how much earlier?
A. I don't know.
Q. Have you ever given the opinion in an individual case, Dr. Nicholson, that a plaintiff who you testified for who had lung cancer, who smoked, would not have gotten asbestos-related lung cancer had they not smoked?
MS. DIX: Objection to the form.
Q. Do you understand my question?
A. I think you are saying that in a joint asbestos exposure cigarette smoking - the person wouldn't have gotten the disease had he not smoked; I don't believe I made such a statement.
Q. On an individual-by-individual basis, would you agree that that statement -- well, let me ask you: That statement being made --
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MS. DIX: Objection to the form of the question.
Q. Do you understand? Let me rephrase it since she's got an objection pending so we are clear.
On an individual-by-individual basis, can you say to somebody who was exposed to asbestos who got lung cancer that it was in fact smoking, not the asbestos, that caused their lung cancer?
A. One can say that the risk attributable to smoking is greater than the risk attributable to asbestos and in most exposure circumstances, but you cannot say you never would have gotten lung cancer had he not smoked; he could have gotten it from the asbestos exposure alone, so it's not an absolute statement.
You cannot make an absolute statement.
Q. And so when you look at it individual by individual and somebody is exposed to asbestos and also smoked, you can't say in fact which one caused lung cancer, can
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you?
MS. DIX: Objection to the form of the question.
A. That's correct. You can only give a risk for each at the time the disease developed.

Q. If we covered this, I apologize, but your work for the Manville trust is limited to your involvement in this case; is that right?
MS. DIX: Objection to the question.
Q. I'm trying to determine whether you did any work for the Johns Manville Trust apart from your involvement as a testifier in this lawsuit.
A. No.
There was the possibility I might do estimates of future cases, but that was set aside.
Q. Have you ever at any point in time in the history of the life of the Johns Manville Trust given the trust any advice on how to allocate risks, for example,

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on payments or disease?
A. No.
MS. DIX: I object to any further line of questioning to the extent it gets into a consultant role that Dr. Nicholson may or may not have played.
Q. You are maintaining as a consultant now for the trust, is that your relationship presently?
A. I guess I am still listed as a consultant with them. I mean I certainly -- certainly have undertaken activities recently, including today.
Q. You said I think in your report, Dr. Nicholson there are no data from which to conduct a time since quit analysis for smoking and asbestos disease; is that right?
MS. DIX: Objection to the form of the question.
Q. You want me to refer you to your report?
A. I am surprised at that. It's not true.

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Q. If you take a look at page 16, fourth line down and we are talking here about parenchymal abnormalities; do you see the sentence that reads, "No data are available that provided information on smoking effects according to time since cessation"?
A. Right, I'm referring to the prior tables, so I apparently looked at that in these articles and made that statement. I mean I don't recall the circumstances, but that may not be universally true, because one could do that in the 2907 data. It hasn't been done by us.
Q. Well, you had the 2907 data, it doesn't say no analysis are done, it is says

no data are available, doesn't it?
MS. DIX: Objection.
Dr. Nicholson answered the question that you posed to him?
A. We are talking about - this paragraph is talking about the data in tables seven and eight. It's very specific to that which has just been presented; starts from the page before and describes numerology, totally

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with those tables.
Q. So whatever Dr. Harris is doing with the 2907, is it fair to say that that hasn't been done yet according to any of the published epidemiology that you are aware of?
MS. DIX: Objection to the form of the question.
A. It wasn't done when I wrote this. I don't think he was even on board.
Q. As far as you know, as of today, hasn't been done in any other epidemiology apart from what Dr. Harris is doing?
MS. DIX: Objection to the form of the question.
A. I can't state that it has not been done in the array of all studies. I believe it has not been done within the studies that proceeded the paragraph where I said it wasn't done, if I read these articles right.
Q. Are you aware as we sit here today of any other studies that provide data that would provide an analysis of the smoking effects according to time since cessation, other than what Dr. Harris is doing?

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A. I am not aware of any.
Q. Fair enough.
Give me a three-minute break.
THE VIDEOGRAPHER: Going off the record it's 4:58 p.m.
(Recess taken.)
THE VIDEOGRAPHER: Back on the record, it's 5:05 p.m.
MR. SCHROEDER: Let's mark this.
(Nicholson Exhibit 7, Chase Kotin article, marked for identification.)
Q. You mentioned, Dr. Nicholson, the Chase Kotin analysis. I want to mark as a copy to your testimony a copy of the Chase Kotin article that you produced to us.
Can you confirm for us, Dr. Nicholson, that Exhibit Number 7 is a copy of the Chase Kotin article that you referred to in your report and your testimony today as it appeared from your file?
A. Yes, it is.
Q. Are you familiar with the 1986 OSHA regulations that addressed the issue of trying to apportion the risk of excess risk

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for lung cancer between asbestos and smoking?
A. I have some familiarity with the 1986 OSHA standard. It's probably federal register. I don't remember that portion of it.
Q. Are you familiar with it enough to be able to answer this question without going through it and you are welcome to go through it.
A. No, I am not. I know the standard used a direct multiplicative model on whatever the underlying risk of lung cancer was and adopted that position. Beyond that, I don't know the details of the discussion.
Q. Let me see if you know this much from it.
MS. DIX: Objection. He testified he didn't know more than --
MR. SCHROEDER: That's fine.
Q. Do you recall enough of it to remember that OSHA adopted analyses that were derivative of something that Dr. Enterline had done that essentially allocated the excess risk almost equally to asbestos and smoking?
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MS. DIX: Same objection.
A. No, I don't recall that.
Q. You are welcome to read as much as you want, I direct your attention to the highlighted portion right there of the 1986 OSHA proceedings?
MS. DIX: I am going to object that you are not showing the witness - I know it's a voluminous document, but he testified he isn't familiar with it.
Q. (Handing.)
What I want to do is direct your attention to that one paragraph that says OSHA's conclusion; do you see that?
A. Yes, I read it and I don't believe that is appropriate.
Q. You would favor the Chase Kotin type analysis?
A. Yes.
Q. So to be clear the view OSHA took in 1986 would apportion more of the excess risk for lung cancer to asbestos than would the model that you have in your report, is that fair?
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MS. DIX: Objection.
A. The Chess Kotin, I believe that is correct, those numbers are higher than are appropriate for most exposures and smoking circumstances.
Q. If I want to compare two groups of

people to do an epidemiologic study, would I want to try to get homogenated between the group?

MS. DIX: Objection to the form of the question.

A. To the extent possible you would like homogeneity on all factors -- on several important factors; age and that that are unrelated to the issue at hand, whatever it might be.

Q. So if I want to compare group one with group two, would I want to compare the risk for the complete group of group one to the complete group of group two or would I compare the risk; would that be better than comparing the risk of the complete group of group one to only the diseased people in group two?

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MS. DIX: Objection to the form of the question.

Q. Do you follow that?

A. What you are looking at is a case control analysis where you are going to take - do a case control analysis and you are looking at the differences in some parameter, that is whatever, XY.

Drink orange juice everyday and look at the prevalence of that parameter and the diseased group compared to a corresponding group of unexposed and without disease and see if there is a significant difference in some parameter that could have affected that disease, so you can --

If you find that everybody with disease was exposed to X, only two percent of the other group was exposed to X, you could contribute X to the disease, so you often make such comparisons.

Q. What I was asking was something a little different and I want to see if you would agree with me.

If I have cohort one that contains

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some diseased and others not diseased, they were exposed to a toxin and then I have cohort two, if it contained diseased and undiseased, everything else being equal, I could compare the two, can I not?

A. You have got two groups, each with disease and each with non-disease.

Q. Correct.

A. Do you have -- are you looking at different exposures of the -- does each group -- are there different exposures to something, to some toxin in each group?

Q. I think you would assume there would be different exposure levels among the group as a whole?

A. Okay, so you have got two groups with different disease and different exposures.
Q. Sure.
A. So you can look to see if that exposure is relevant.
Q. And so I would want to compare the groups and their exposures as a whole opposed to picking out just the diseased people in a

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group and comparing them with the other group as a whole?
MS. DIX: Objection to the form of the question.
A. In a cohort study, that's what you would want to do, but at other times you may simply select the diseased people, as I stated and you may want to do a distribution of age different; you may do it age standardized, so there is ways to deal with other factors that play a role, but are unrelated to the toxin you want to look at, but in general you want to compare one whole group with the other, as I understand the hypothetical circumstances that you posed.
Q. If I compare only the diseased in one group and their rates or their relative risks, if you will, to the cohort, the whole other group - you follow me?
A. The diseased has a relative risk of one. They are all diseased and so it doesn't make sense to compare that group's risk with the group that has some disease and some not, that sets up a nonofficial

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circumstance as you described it.
Q. So let me make sure I follow that, if I have group B and it's all diseased by definition and I compare them with a cohort, some of whom are diseased and some of whom are not, are you saying that that is an appropriate or not an appropriate comparison?
A. On a cohort basis it's not appropriate to say this is a hundred percent disease and this is five percent.
Q. Why is it inappropriate?
A. Because it's arbitrarily set up, an artificial group. You can do it on a case control basis and look at the differences in the exposures that might have taken place, so you can do it on that basis, but if you are setting up a cohort analysis where you are going to compare whatever average exposure they had with the average exposure in the other one and one is all diseased and one is not, you set up an artificial circumstance.
Q. I have one last question for you, Dr. Lilis' 1991 paper I think if I remember correctly you told us was the paper that

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addressed the 2907 insulators; did I get that right?
A. I'm not sure.
MS. DIX: Objection.
MR. SCHROEDER: Let's mark this as 8.
(Nicholson Exhibit 8, Lilis' report, marked for identification.)
Q. Exhibit 8, can you tell us that that is in fact Dr. Lilis' 1991 study?
A. Yes, it is, on the 2907.
Q. That's the same group that you gave the data to Dr. Harris?
MS. DIX: Objection to the form of the question.
A. Yes.
Q. I would direct your attention to page six, below the figure, I would ask you to read that paragraph and tell me whether in fact Dr. Lilis concluded that no smoking effect was found in this group for pleural abnormalities?
A. For pleural abnormalities?
Q. Yes.
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MS. DIX: Objection, the document speaks for itself.
A. Yes, she states that there was no detectable effect in history of cigarette smoking for pleural calcifications.
Q. In fact she says on the sentence that comes from the page just prior she speaks to pleural thickening?
A. She states here also that smoking status did not effect the prevalence of pleural fibrosis.
Q. Right. Above doesn't she say the prevalence of pleural abnormalities did not differ significantly in the history of those with cigarette smoking opposed to those who never smoked?
MS. DIX: Objection.
A. Yes.
Q. Based on your training and education and knowledge of these studies, Dr. Lilis is concluding at least in this study that she did not observe any smoking effect on pleural abnormalities?
MS. DIX: Objection, the document
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speaks for itself.
Q. You can answer the question.
A. That was her conclusion for that particular analysis.
MR. SCHROEDER: I am done.
MR. KAZAN: I have a few

questions.

MR. SCHROEDER: If you are going to ask, I am going to object because Ms. Dix had been proffering all the objections. I will object to any line of questions.

MR. KAZAN: Your objection is noted and overruled. Do we have time on the camera?

THE VIDEOGRAPHER: You got two minutes left.

EXAMINATION BY
MR. KAZAN:

Q. Doctor, you were asked some questions about the green sheets; do you have personal knowledge as to whether or not insulators in fact did or did not read or study the green sheets that they received in

their magazines?

MR. SCHROEDER: Objection to the form of the question.

A. I have no knowledge of their reading of it, what fraction read and what fraction did not. I made the joke that they may not have read it.

Q. Doctor, you first came to work at Mount Sinai in 1969 according to your CV; is that correct?

A. Yes.

MR. SCHROEDER: Objection to the form of the question.

Q. From your review of the literature, when did Dr. Selikoff first began doing any work with the insulators in the New York and New Jersey area?

MR. SCHROEDER: Objection to the form of the question.

A. I know it certainly was well before 1964. I can't tell you specifically the first date, but he had already published a mortality study done with the cooperation of the union in 1964.

Q. Do you have any personal or firsthand knowledge of the extent to which, if any, Dr. Selikoff had anything to do with attempting to educate union workers in the New York and New Jersey areas or anywhere else about the potential hazards of asbestos and/or smoking in the years prior to your arrival at Mount Sinai and specifically with reference to the early 1960s?

MR. SCHROEDER: Objection.

A. I don't know of his work in that period of time.

Q. One-way or the other?

MR. SCHROEDER: Objection.

A. One way or the other. If he met

with a group I would imagine he would have certainly warned of the hazards of asbestos and also smoking; he certainly was aware of the separate risk of smoking.
MO MR. SCHROEDER: Wait, object, move to strike as not responsive.
Q. Doctor, you don't know of your own knowledge one way or the other what, if anything, Dr. Selikoff did or didn't do with

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those workers in the years prior to 1969?
A. That's correct.
MR. KAZAN: Thank you, no further questions.
THE VIDEOGRAPHER: Going off the record it's 5:23.
(Recess taken.)
THE VIDEOGRAPHER: Back on the record 5:27.

FURTHER EXAMINATION
BY MR. SCHROEDER:
Q. Dr. Nicholson, who would be aware of what activities, if any, Dr. Selikoff engaged in in the 1960s with respect to whether or not he warned any union members?
A. I don't know a person that would be appropriate to answer that. Hammon might know it, but he's dead.
Q. Are there any documents to your knowledge that would address that issue?
A. No, not to may knowledge.
MR. SCHROEDER: Thank you, sir.
THE VIDEOGRAPHER: Going off the record, this is the conclusion of tape number 3, it's 5:27 p.m.
(Time noted: 5:27 p.m.)

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A C K N O W L E D G M E N T

STATE OF NEW YORK)
:ss
COUNTY OF)

I, WILLIAM J. NICHOLSON, Ph.D., hereby certify that I have read the transcript of my testimony taken under oath in my deposition of June 5, 2000, that the transcript is a true, complete and correct record of my testimony, and that the answers on the record as given by me are true and correct.

_____
WILLIAM J. NICHOLSON, Ph.D.

Signed and subscribed to before me, this dayof , 2000.

_____
Notary Public, State of New York

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C E R T I F I C A T E

STATE OF NEW YORK)
) ss.:
COUNTY OF NASSAU)

I, CINDY DILEONARDO, a Notary

Public within and for the State of New York, do hereby certify:

That WILLIAM J. NICHOLSON, Ph.D., the witness whose deposition is hereinbefore set forth, was duly sworn by me and that such deposition is a true record of the testimony given by such witness.

I further certify that I am not related to any of the parties to this action by blood or marriage; and that I am in no way interested in the outcome of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand this 7th day of June, 2000.

CINDY DILEONARDO